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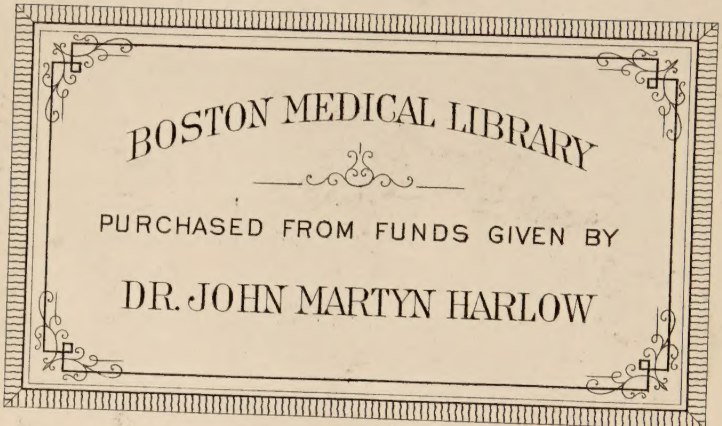


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PERITONITIS



J. GARLAND SHERRILL



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SURGICAL MONOGRAPHS

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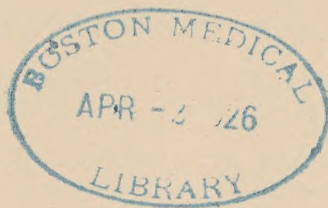


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PREFACE

In offering this work to the profession an effort has been made to eliminate as much as possible unessential subject matter and to present only material of practical interest. The style may be somewhat dogmatic, but I have endeavored not to become pedantic in expression.

There are many things in this subject which are likely to produce a difference of opinion. This is so much the case that for more than a quarter of a century it has furnished the material for many a surgical debate.

What is presented in these pages represents what I believe to be the best surgical opinion. The methods given are those found the most satisfactory by the writer, and the work is submitted with the hope that some benefit may result from the perusal of it.

J. GARLAND SHERRILL

LOUISVILLE, KY.

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PERITONITIS

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CHAPTER I

THE PERITONEUM

ANATOMY

The lining membrane of the abdominal cavity is formed of two layers: a basal layer (*tunica propria*) composed of areolar and connective tissue, and a free surface of endothelial cells derived from the mesothelium. The basal layer carries lymphatic vessels and blood-vessels for its nourishment, and also permits the passage between its folds of both blood and lymph vessels to the respective viscera. The basal structure is attached to the parietes for a considerable extent, the remainder being attached to and forming the external covering of the viscera. One large fold of reduplicated peritoneum makes up the structure of the omentum, while that portion of the peritoneum which extends from the viscera to the abdominal wall makes, by the contact of its two basal structures, a peritoneal ligament covered externally on each surface by the endothelial structure. These ligaments are mesocolon, mesentery, etc., depending on the organ to which they are attached.

The peritoneal cavity is a closed sac in the male, while in the female the patency of the distal end of the lumen of each fallopian tube permits a communication with the external air. The peritoneum is a serous membrane, and in health a small amount of serous fluid moistens its surface, affording lubrication which permits mobility of the intestines and prevents their adhesion to other organs. In disease this surface becomes dry, glazed, roughened, and adhesions at points of contact frequently result. This closed bag or sac is placed about the viscera very much as if one had taken a silken bag and pushed it into all the crevices.

It would be more proper to describe this as an ingrowth of the viscera, pushing them forward into the peritoneal structure, which more or less completely surrounds them. Strictly speaking it is a fallacy to state that a structure is intraperitoneal (within the peritoneal cavity) and redundant to say extraperitoneal (outside the same), but the use prevails, nevertheless, to express in relative terms the extent to which a structure is enclosed by the peritoneal folds.

It matters not from what point one starts to trace the peritoneum; the same point can always be reached again without interruption. If the tracing begins on the upper surface of the liver and continues to the upper margin of its posterior border, it will be found that the peritoneum ascends from the liver to the diaphragm and passes forward over the inferior surface of that structure to the anterior parietal wall. It can then be traced down the anterior parietal wall to reach the umbilicus, at which point the remains of the artery lying under the peritoneum becomes the round ligament of the liver and the peritoneum continuing across this forms in its upper portion the so-called falciform ligament of the liver. This structure is of importance to the surgeon in operations in the upper portion of the abdomen. From the umbilicus the membrane can be traced along the parietes to the pelvis. Lying immediately in front of the peritoneum in this region are the remains of the urachus, a prominent cord extending from the fundus of the bladder to the umbilicus. A short distance on each side and forming a triangle on each side with the urachus, the remains of the hypogastric arteries of the fetus are found. In close relation with the course of these cords are the deep epigastric arteries, which come off from the external iliac and course for some distance between the peritoneum and the transversalis fascia before entering the sheath of the rectus muscle, in which they ascend to anastomose with the internal mammary (superior epigastric) arteries from the subclavian. These arteries are accompanied by their vena comites.

The urachus, the hypogastric cords, and the epigastric arteries make three peritoneal folds in the hypogastrium, thus forming two triangles, base down, resting on the pubes, with the apices lying together at the umbilicus. Between these folds lie two peritoneal pouches and outside of the epigastric arteries are two additional pouches, which are thought to be of some importance in the development of hernia in the inguinal region. The structures above described are extraperitoneal, and the peritoneum lying over them makes the pouches. When the peritoneum is followed down the anterior abdominal wall along the urachus, the fundus of the bladder is reached. The distance of this point from the navel varies greatly, and is dependent largely upon the amount of distention of the bladder. It may reach almost to the umbilicus in the overdistended organ, or may lie below the pubic arch in case it is empty. The peritoneum is very loosely attached to the abdominal wall, from the fundus of the bladder to the pubic bone, and the properitoneal space of Retzius is filled with a considerable amount of loose fatty tissue, which permits the changes in size of the bladder with the least discomfort to the patient.

The surgeon must always bear in mind the fact that in many individuals, and particularly in children, the bladder when distended reaches far above the pubic bone, and measures must be taken to prevent its injury in median abdominal sections. It is also true that in some pathological conditions, as fibroid of the uterus, or cyst of the ovary, the empty bladder may ride very high, being drawn upward as the growth progresses, and is the more liable to injury.

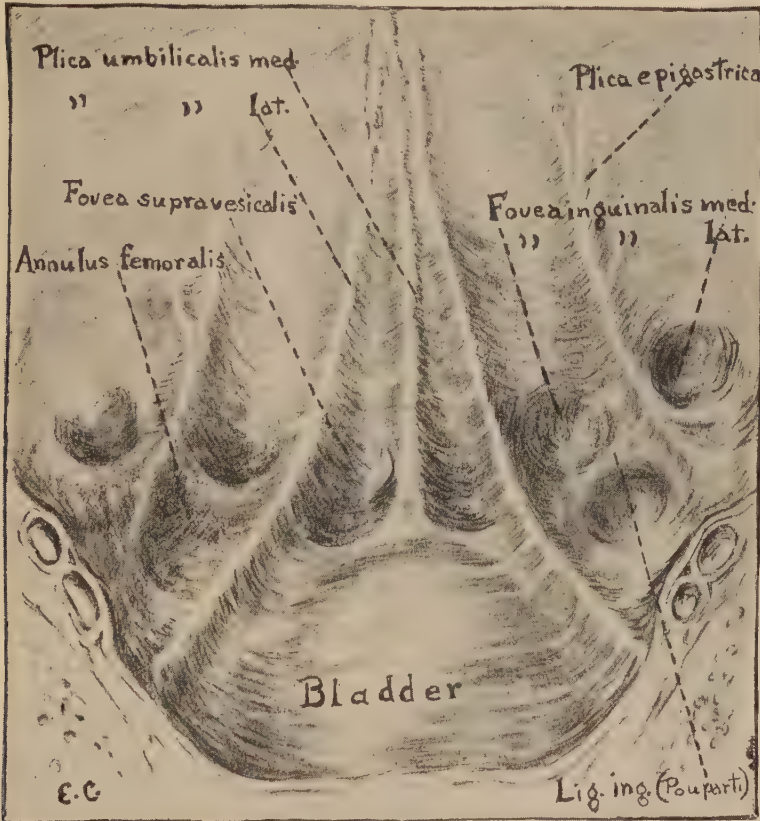


FIG. 1.—FOLDS AND FOSSAE OF THE ANTERIOR ABDOMINAL WALL. (Redrawn from Waldeyer.)

In the female the peritoneum passes over the fundus of the bladder to the point of junction of this organ with the uterus. It then passes over the anterior surface of the uterus and the adnexæ, covering the round and forming broad ligaments. The broad ligament reaches across the pelvis in the perpendicular plane from the uterus to the bony pelvis. It is formed by a duplicature of the peritoneum as this is reflected from the anterior and posterior surface of the uterus. Within the broad liga-

ment are carried the ovarian and uterine arteries and veins. Posterior to the broad ligaments lie the ovaries with the oviduct enclosed by its upper border on each side. These structures receive a complete peritoneal covering except at the fimbriated end of the oviduct where the lumen is open and there is a communication of the peritoneal surface with the external air.

The peritoneum continues over the fundus and posterior surface of the uterus and extends backward on each side to the sacrum, forming the uterosacral ligaments on each side. Between these two ligaments the peritoneum dips down and is attached to the upper portion of the rectum. In this region there remains a considerable fossa, the pouch of Douglas. The peritoneum continues up the posterior wall from this point as it does in the male.

The peritoneum can be traced over the bladder in the male, and the spermatic ducts may be observed coursing under its structure from the external rings upward over the bladder to its fundus and down its posterior wall to the bas fond, where they proceed forward to join the duct of the seminal vesicles and form the ejaculatory duct on each side.

These structures are of surgical importance, particularly in genital tuberculosis, which may spread along the duct from the testicle, and may even infect the overlying peritoneum, although this must be of rare occurrence. Operations for removal of the tuberculous testicle and for sarcoma of that organ require wide removal of the spermatic duct, which can be accomplished easily without invading the peritoneal cavity. From the base of the bladder the peritoneum extends backward across the pelvic floor to the middle third of the rectum, lying over the rectovesical folds at the side of the gut. It passes laterally, covering the lateral walls of the pelvis, and covering the ureter and the large vessels and nerves of the pelvis, and covers the iliac fossa on each side, being separated from the muscles in this region, the psoas and the iliacus, by the iliac fascia, while in the pelvis the pelvic fascia lies between the serous structure and the vessels. Both of these delicate fasciæ are continuous with the transversalis fascia in front and the lumbar behind.

The peritoneum may be traced upward along the anterior wall of the rectum for some distance, only covering its anterior and part of its lateral region, but in its upper portion it entirely surrounds the rectum and forms the mesorectum, up to the brim of the pelvis. Here the attachment of the sigmoid to the pelvic wall is very close and posteriorly is not covered with peritoneum, being without a mesentery. The sigmoid is for the greater portion of its extent entirely covered with peritoneum and has a rather long mesosigmoid. The upper end of the sigmoid is

attached closely to the wall at the sacro-iliac articulation, and only about two or three inches from the attachment of its extremity. It is doubled back, therefore, somewhat in the shape of the letter S and it is because of this arrangement that it derives the name sigmoid. On each side of the abdomen the peritoneum covers the colon, both the right or ascending and the left or descending colon being only partially covered with this structure. The caput coli, in some instances, is entirely surrounded with peritoneum, while in other individuals it is only partially covered. The appendix is usually covered throughout with peritoneum, and has a well-developed mesentery. The latter, however, varies much in different individuals. The appendix in some cases, particularly where the caput coli has little mobility, is sometimes found behind the cecum, lying extraperitoneally.

There are quite a number of errors in development in connection with the caput coli, which, with its appendix, becomes a most important consideration for the surgeon. In the ileocolic fold there is a pouch in which hernia sometimes occurs. The peritoneum traced up the posterior wall covers entirely the jejunum and ileum, each having a distinct mesentery, in and beneath which lie a large number of lymphatic glands, which are important in the process of absorption, and which are often the site of inflammatory processes. Sometimes these processes follow and sometimes precede inflammation of the peritoneum.

The mesentery of the small bowel extends from the origin of the superior mesenteric artery from the abdominal aorta, just opposite the second lumbar vertebra, and extends in a slightly curved direction, convexity to the left, to the right sacro-iliac junction. This distance, less than five inches, makes the root of the mesentery, while the intestinal attachment is twenty or more feet in length, making the mesentery fan shaped and causing the intestine to lie in coils. This arrangement permits the free movement of the coils upon each other, but from the short base of the mesentery it tends to prevent torsions, not, however, always successfully.

At the upper end of the jejunum, where it joins the lower end of the duodenum behind the peritoneum, and seen when the omentum and transverse colon are lifted, the jejunum is supported by a peritoneal band, the ligament of Treitz, a structure of importance in the operation of posterior gastro-enterostomy. Here also there sometimes occurs an internal hernia.

Passing upward from the ligament of Treitz, the peritoneum forms the mesocolon in conjunction with the posterior layer from the lesser peritoneal sac and passes forward to surround the transverse colon.

From this point two layers pass forward and downward; returning, the two layers pass upward to the stomach, forming the great omentum. The four layers are fused together in the adult, but in some young individuals the peritoneal space may be detected within its folds. The two upper layers pass to the stomach and surround it, one above and one behind, and being joined together posteriorly, make up the lesser omentum, which passes up to cover the lower surface of the liver, and

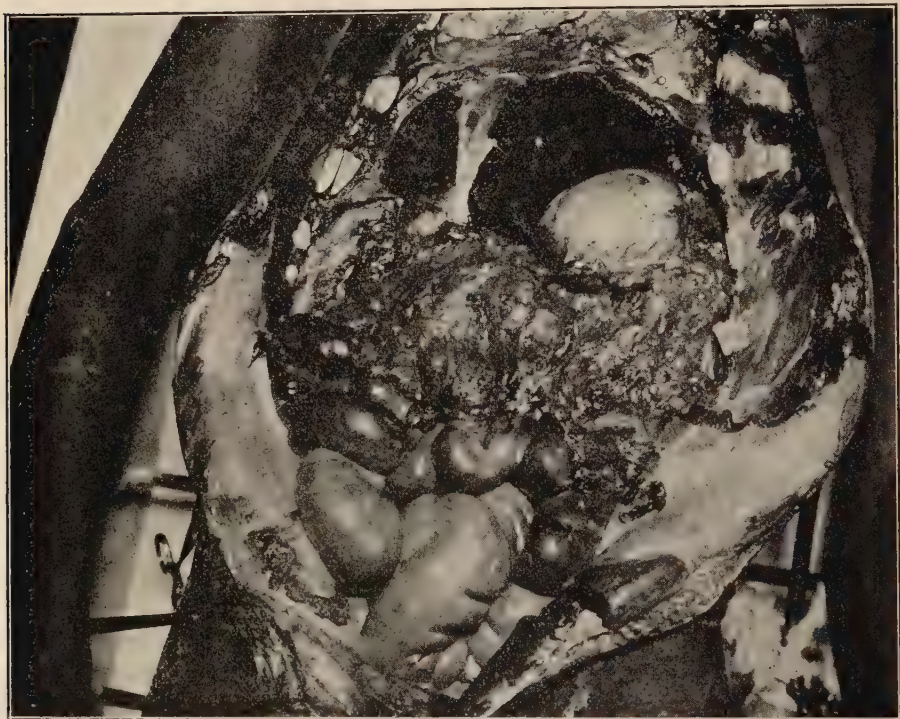


FIG. 2.—NORMAL PERITONEAL SURFACES.

Note the liver, falciform ligament, gall-bladder, stomach, great omentum, intestines, and parietal peritoneum.

pass downward over the posterior abdominal wall, forming the posterior limits of the lesser peritoneal sac. The right border of the lesser omentum carries between its folds the hepatic duct, the hepatic artery, and the portal vein to the liver. They lie from right to left and from before backward—duct, artery, vein—a matter of considerable importance to the surgeon in operation upon the biliary passages and the duodenum.

The left portion of the lesser peritoneal sac follows the posterior abdominal wall to form one of the splenic ligaments, and joining the visceral layer extending from the posterior surface of the stomach forms

the gastrosplenic omentum or ligament and surrounds the spleen, passing to the parietal wall at the diaphragm.

At the ligament of Treitz, the duodenum lies behind the posterior peritoneum, and only in the upper portion of the second or descending part of the duodenum does the peritoneum cover front and sides, while the first portion of the duodenum is, like the stomach, fully surrounded by the peritoneum. The pancreas lies entirely behind the peritoneum, in close relation at its head with the second portion of the duodenum, and its duct empties into this intestine, either with the ductus communis choledochus, or through a separate opening (Wirsung, Santorini). The portal vein and the superior mesenteric artery pass through a notch in the upper border of the pancreas near the duodenum, while the splenic artery and vein lie on the upper border of the pancreas as they extend to the spleen. A large number of vessels, called the vasa brevia, lie in the gastrosplenic fold, and may be the source of much hemorrhage in splenectomy; hence they should be doubly clamped and ligated, as well as the splenic artery and vein. The arrangement of the vessels about the duodenum and the pancreas accounts to a large degree for the rotation of the intestines, which takes place in development of the fetus, and also for the permanent position of these structures and of the transverse colon as well.

It is well for the abdominal surgeon to remember that in gunshot wounds of the mesentery, pressure upon the vessels at its root will control serious hemorrhage until the local injury can be found and clamped, also that by grasping the foramen of Winslow and pressing upon the lesser omentum, serious hepatic bleeding can be controlled temporarily. On each flank the peritoneum passes backward from the colon to cover the renal space in front.

Beneath this structure the kidney lies under a thin, fibrous capsule of fascia, continuous with the subperitoneal fascia, which passes over its anterior surface, but which has an interruption at its lower extremity, a few fibers being thickened into an arch, under which kidneys with long vascular pedicles sometimes escape and become so-called floating or wandering kidneys. This anterior layer of fascia at the external border of the kidney is connected with the fascia lying behind the kidney and is continuous with the transversalis fascia.

The Fascia perirenalis, or Gerota's Capsule.—This layer of fascia was described by Gerota, and it receives his name. The capsule is developed to a greater degree upon the left side. It consists of a portion of the lumbar fascia which splits at the external convex border of the kidney into two layers. The anterior layer, more delicate than the pos-

terior, extends across the front of the kidney and is attached to the retro-peritoneal fascia behind the mesocolon. It passes over the anterior surface of the pancreas and crosses the anterior surface of the vessels lying in front of the spine, where it is continuous with the like structure on the opposite side. The posterior layer of this fascia extends upward to the fascia under the diaphragm and also to the sheath of the psoas muscle below, being attached to the intervertebral disks and vertebrae. Above the kidney the two leaves enclose the suprarenal gland, fuse together, and are attached to the diaphragm. The capsule is open at its lower portion and to some extent on the inner side, somewhat like a flowing sleeve. It permits the kidney to move up and down upon the axis of its vessels somewhat like a pendulum. When this motion exceeds thirty degrees it becomes abnormal. In addition to the freedom of mobility within the capsule of the kidney, this fascia determines, also, to some extent the direction taken by suppurative inflammatory exudates into the perirenal space. It has also some function in preventing suppuration about the kidney from extending forward and involving the overlying peritoneum, because it tends to limit such extension.

Underneath this fibrous tissue lies a thick layer of loose, fatty tissue, and under this the fascia proper of the kidney.

The gall-bladder is covered for a large portion of its extent with peritoneum, but a portion lies in direct contact with the liver tissues. The absence of this peritoneal covering accounts for those cases in which gall-stones ulcerate into the liver, the wall of the gall-bladder without its protective peritoneum being broken down from pressure, and the stones are found in an hepatic abscess.

Outside of the peritoneum a thin but relatively strong layer of fascia lines the entire abdominal region. At some points this fascia is closely attached to the peritoneum, while in other regions, notably in the prevesical space, in the renal and lumbar region behind the colon on each side, there is a quantity of loose cellular tissue. In such spaces there is a marked tendency to permit the spread of suppurative infections. The writer has observed infections of postcecal appendicitis extend behind the colon and right kidney to the subdiaphragmatic space, and drain by rupturing through a bronchus, with recovery.

HISTOLOGY

This subject has received an enormous amount of study, which indicates the importance of a knowledge of the structure and function of the peritoneum. It is only through such a study that one can under-

stand the physiologic processes of absorption and the method by which pathogenic material is disposed of under normal conditions.

A very strict interpretation of this structure from the histologist's standpoint would result in the conclusion that it consists of two layers. One of these, on the free surface, is composed of a single layer of

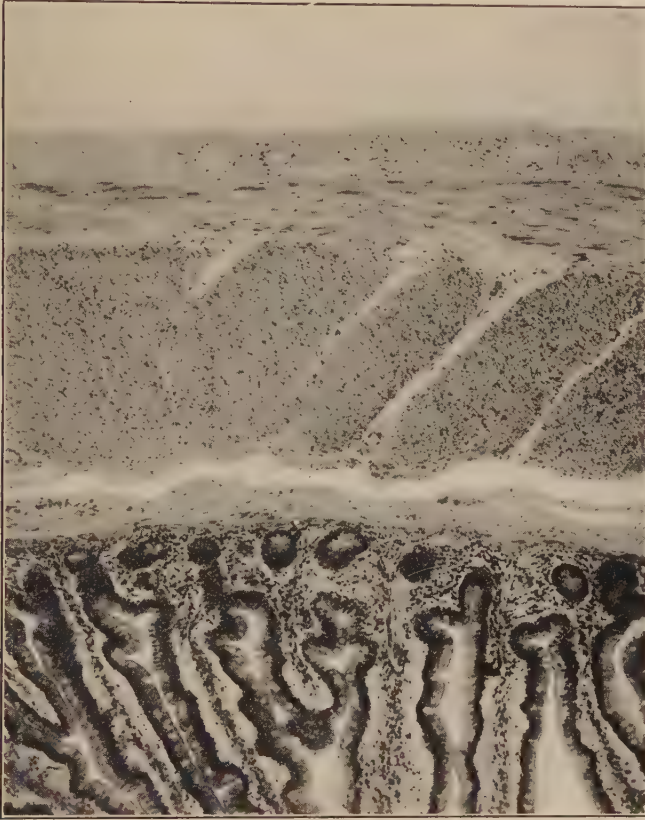


FIG. 3.—HUMAN SMALL INTESTINE. NORMAL PERITONEUM. PHOTOMICROGRAPH 80 DIAMETERS.

Upper layer serosa, blood-vessels, and lymphatics. Outer longitudinal muscle. Intermuscular layer. Circular or inner muscular layer. Submucosa. Mucosa. Intestinal glands. Crypts of Lieberkuhn. (Collection of Prof. S. I. Kornhauser, Department of Anatomy, University of Louisville.)

polygonal, flattened endothelial cells. Beneath it the second layer consists of the pavement membrane, on which these endothelial cells lie. This does not include the connective tissue structure which carries the vessels to their respective organs.

In reality, therefore, there are three layers to be considered as forming the peritoneum. In certain locations the connective tissue is some-

what thickened, as in the peritoneal ligaments of the liver, the phrenocolic ligaments, the broad ligaments of the uterus, etc.

The endothelial covering of the free surface of the peritoneum is shown under the microscope to be composed of polygonal, pavement epithelial cells presenting a smooth glistening surface. These cells are somewhat irregular in shape, and are held together by very delicate homogeneous cement substance. This cement substance between the cells is regarded by some as a fluid similar to lymph, while others, notably



FIG. 4.—NORMAL PERITONEUM, HIGH POWER 335 D, OF SAME SPECIMEN.

Simple pavement endothelium of serosa. Serosa, blood and lymph vessels. Sub-serosa. Longitudinal muscle. Intermuscular layer. Circular muscle. (Dr. S. I. Kornhauser.)

Flemming and Merk, observed that this substance differed from the fluid in the lymph vessels.

The endothelial cell itself varies considerably in size, from 20 to 50 or more microns. It can be distinguished readily from the ellipsoid cells of the blood and lymph vessels by its form alone. The cell contains a large nucleus measuring from 10 to 16 microns,⁵ usually placed near the center of the cell, with one or two nucleoli. The protoplasm is transparent in the fresh state. When stained it is finely granular.

Some observers have claimed to find cilia upon these cells. It is likely that these supposed cilia are in reality fibrils resulting from coagulation

of fluid normally covering the cells. This is perhaps one of the early changes in the formation of adhesions.

Some of the older writers claimed to find stomata between the cells which were supposed to permit the passage of material which might be present in the peritoneal sac. The views of His⁶ upon the subject were generally accepted and Cohnheim² made the presence of stomata and stigmata in the blood-vessel the basis of his theory of inflammation. Some of the later writers, notably Auerbach¹ and Klein,⁹ held to the opinion that the openings believed to be stomata were artificial products. One of the strongest arguments against their presence was that of Ranvier, who showed that when the surface to be treated with nitrate of silver was first rinsed with water their number was much reduced.

There seems to be this decided divergence of opinion because of different methods of technic in staining these cells. It would appear that the proper way to make such studies would be in living tissue. Under such circumstances the cell would retain to a considerable degree its normal function. In as much as every cell has some power of contraction in response to an irritant, the thought arises: Can not the normal cells by contraction of their protoplasm provide an opening which may not otherwise be seen, and may not the contentions of both groups be somewhat harmonized?

Those who follow the arguments of Muscatello are inclined to coincide with his views that there are no stomata but that the openings, so considered, are either the result of badly prepared specimens or optical illusions.

Blood and Lymph Vessels of the Peritoneum.—At the root of the mesentery the peritoneum simply affords a covering to the blood vessels. As the vessels proceed through the mesentery they become more closely associated with it and near their terminal divisions this association becomes intimate.

These blood-vessels for the most part supply the structure covered by the peritoneum, only a small portion going directly to supply the membrane. The very marked vascularity of the subjacent membrane, however, takes care of a considerable portion of the nourishment of the basement cells.

The mesentery is supplied with numerous vascular channels which are only called into use in case of necessity, but under irritation they become filled with blood.

The lymphatic vessels of the peritoneum lie beneath the basement membrane, those over the hollow viscera emptying into the mesenteric radicles, while those over the parenchymatous organs join those of

the parietal peritoneum and become a part of the general lymphatic system.

Throughout the alimentary canal two sets of lymph vessels are found, one lying in the mucosa including the submucosa, and the other in the muscularis.

"In those structures supported by duplicature of serosa (mesenteries) the finer network of the mucosa proper extends to a coarse and charac-

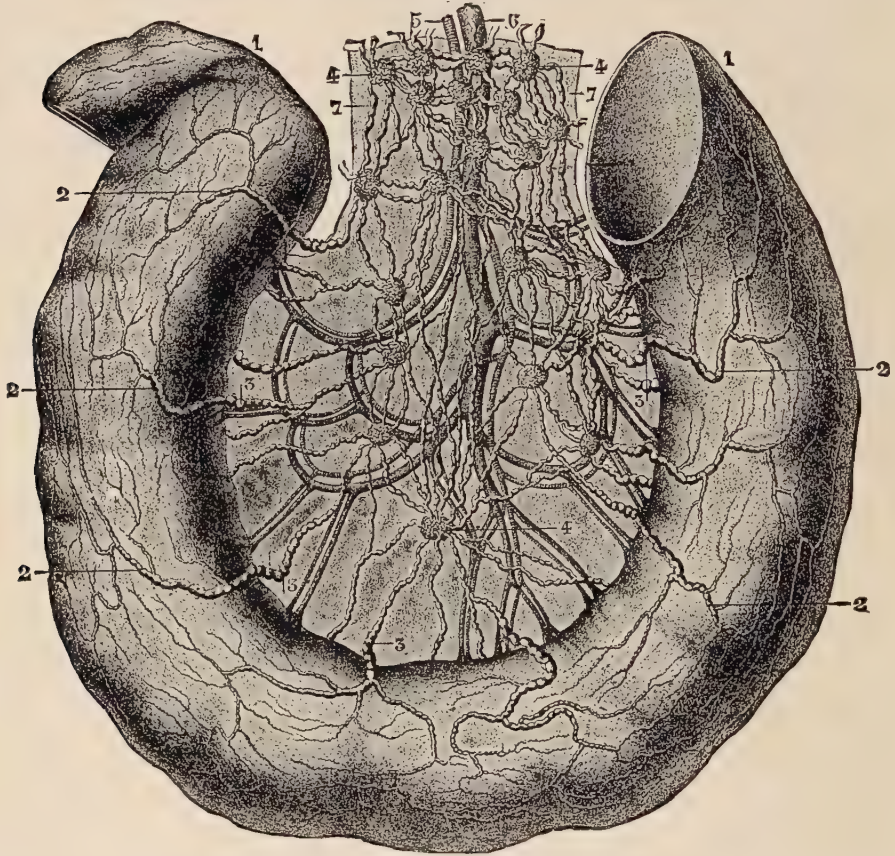


FIG. 5.—LYMPHATICS OF PERITONEUM. MESENTERIC LYMPH NODES. (After Sappey.)
From *Reference Handbook of Medical Science*, Fig. 3868, 1916, Vol. VI, p. 174.
(Courtesy of Wm. Wood Publishing Co.)

teristic network in the submucosa, finally the connecting trunks penetrate the wall at the attached edge and join the lymphatics of the muscularis. The lymphatics of the muscularis are found throughout its entire thickness, but soon become subserous and wind around the attached edge and with those of the mucosa extend between the layers of the supporting membrane, mostly in company with the blood vessels to lymphatic

nodes which are also situated between the serosal walls of the mesentery." ¹⁰

The lymphatics of the small intestines receive the name lacteals because during digestion they carry a milky substance. These vessels enter a group of mesenteric lymph nodes and are continued upward through two or more additional sets before emptying into the receptaculum chyli, being joined by the efferent vessels from the celiac-mesocolic nodes.

There is a wide communication between the lymphatic channels throughout the abdominal and pelvic cavity, which anastomose with those of the parietes of the abdomen. This explains in some degree why the peripheral nodes are often involved in deep malignant growths, and the latter should be carefully investigated when such condition is suspected.

The absorptive power of the peritoneal lymphatics is very marked and so is that of the blood-vessels. The question of which vessels carry on the larger portion of absorption remains undetermined and from a clinical standpoint it would seem to make but little difference since it is well known that absorption is exceedingly rapid.

According to Johnson ⁸ from experiments at Cornell University Medical School it was shown that blood cultures taken five minutes after the injection of virulent cultures of pyogenic organisms into the peritoneum showed the presence of these organisms in the blood. All methods of dealing with peritoneal infections must take into consideration the rapidity of absorption from this cavity.

The Lymphatic Trunks.—The development of the lymphatic system has received much study, particularly in reference to its relation to the connective tissue spaces, but up to the present time the moot points are not fully settled. Two principal views concerning the development of the lymphatic apparatus have been offered which are apparently irreconcilable.

One contention is that the lymphatics develop as mesenchymal spaces in the embryonal connective tissue. These primary vessels according to this view obtain their endothelial lining from the mesenchymal cells bordering the spaces at their first appearance. These vessels are not developed from the blood-vessels, but join these structures later. Junction with the veins is effected through an intermediate venous element with which they connect. Huntington and McClure ⁷ hold this view as the result of extensive studies on animals.

To Sabin ¹¹ must be credited the vigorous support of the opposite view that the entire lymphatic system is derived from outgrowths from the blood-vessels. The lymph vessels are outgrowths from the blood-vessels at certain distinct points and are continuous from these points,

their growing ends appearing as blind tubes. The tissue spaces and lymph spaces, according to this view, are entirely distinct, as is the case with the blood-vessels.

That such budding may occur from the endothelial lining of the blood-vessels must be admitted from the new vascular formations which occur during the process of normal repair. The same property of this endothelial tissue to proliferate is noticed in certain neoplasms.

According to Gage³: "The problem of the ontogenesis of the lymphatic system is that of its endothelial lining.

"It should also be stated that once formed the vascular endothelium—both in the blood and lymph vascular systems—possesses a marked power of growth, showing itself in the extension of channels, regeneration after destruction of definite areas and pathologically (entotheliomata).

"The difficulty, therefore, that attends an understanding of the development of the lymphatic system lies in the interpretation of the first appearance of the endothelium."

The large lymphatic vessels which drain the lymph from the radicles of the various portions of the body appear as two distinct trunks, the ductus lymphaticus dexter and the ductus lymphaticus sinister, or thoracic duct. In man and the higher animals there is a marked difference in the length of these two structures and in the extent of tissues which they drain. In the lowest mammals these structures are more nearly equalized both in extent and in function. In the very lowest animals they are both equal and symmetrical.⁴

The right lymphatic duct in man is quite short in extent, about fourteen millimeters long, while its diameter is about the same size as the thoracic duct. It receives the accumulations of lymph from the right side of the head, neck, right arm, right sternal, mediastinal, and bronchial plexuses. It empties into the subclavian at its junction with the internal jugular. At this point the duct may be divided into a number of channels which open separately into the blood-vessel. In some cases no distinct single duct is present on this side, but a number of confluent ducts open directly into the vein.

The more important lymphatic trunk and particularly as related to drainage of the peritoneal region is the thoracic duct which empties into the vein in a manner similar to the right duct at the left angulus venosus. This duct collects the lymph from the lower extremities, the pelvic structures, the genital and urinary organs, the digestive organs including the alimentary canal, the pancreas, a considerable portion of the liver, the spleen, the diaphragm, the left half of the trunk above the navel and a portion of the right side of the thorax. The distal portion of this trunk

lies in front of the vertebra at about the level of the first or second lumbar, where the right and left lumbar ducts coalesce.

Another trunk (the azygos) joins in the formation of the receptaculum chyli or cisterna chyli, which is in reality the beginning of the thoracic duct. This may sometimes be absent as a distinct cistern, its place being taken by a network of vessels. The length of the duct varies with the conformation of the individual, the averages being about forty-two centimeters. Its diameter is between two or three millimeters and is larger below.

Occasionally two trunks of considerable size pass upward and join each other in the chest. A very considerable anastomosis of the lymphatic vessels is usual. The thoracic duct passes with the aorta between the pillars of the diaphragm lying for the most part to the right of the aorta, finally crossing posterior to that artery and crossing the spine in a gradual curve, at about the third to the sixth thoracic vertebra. It curves to the left and forward between the vertebral vessels and the jugular vein to its point of junction with the internal jugular and subclavian vein. There is an abundant supply of valves in the lymphatic ducts. The walls of the vessel are neither so distensible nor so strong as those of the veins. The intravascular pressure is not so high.

The lymphatic vessels from the leg, thigh, buttocks, and a portion of the loin are connected with the lymph nodes in the inguinal region. These nodes appear as two groups, superficial and deep. The latter receive the lymph from the superficial inguinal glands and also the deep channels of the thigh and the lateral portions of the pelvis.

The superficial lymphatics of the abdominal wall below the level of the umbilicus reach the thoracic duct after passing through the superficial and deep inguinal glands. The lymphatics of the testicle follow the spermatic vessel and join the lumbar nodes. These vessels are strikingly easy to inject.

The vessels of the seminal vesicles reach the iliac nodes through two trunks. Those of the prostate are collected into two trunks on each side, one of which goes directly to the hypogastric nodes, while the other passes around the bladder to enter a hypogastric node from the side.

A portion of the lymph vessels from the female genitalia, the lower third or fourth of the vagina and the pudendum reach the inguinal lymph glands, while the others connect with the hypogastric and inguinal nodes. The lymphatics from the kidney and the adrenal reach the lumbar nodes.

The intestinal lymphatics reach the receptaculum through the mesocolic nodes and the lumbar (aortic) nodes. Those about the arms pass with the superficial vessels of the skin to the inguinal glands. The

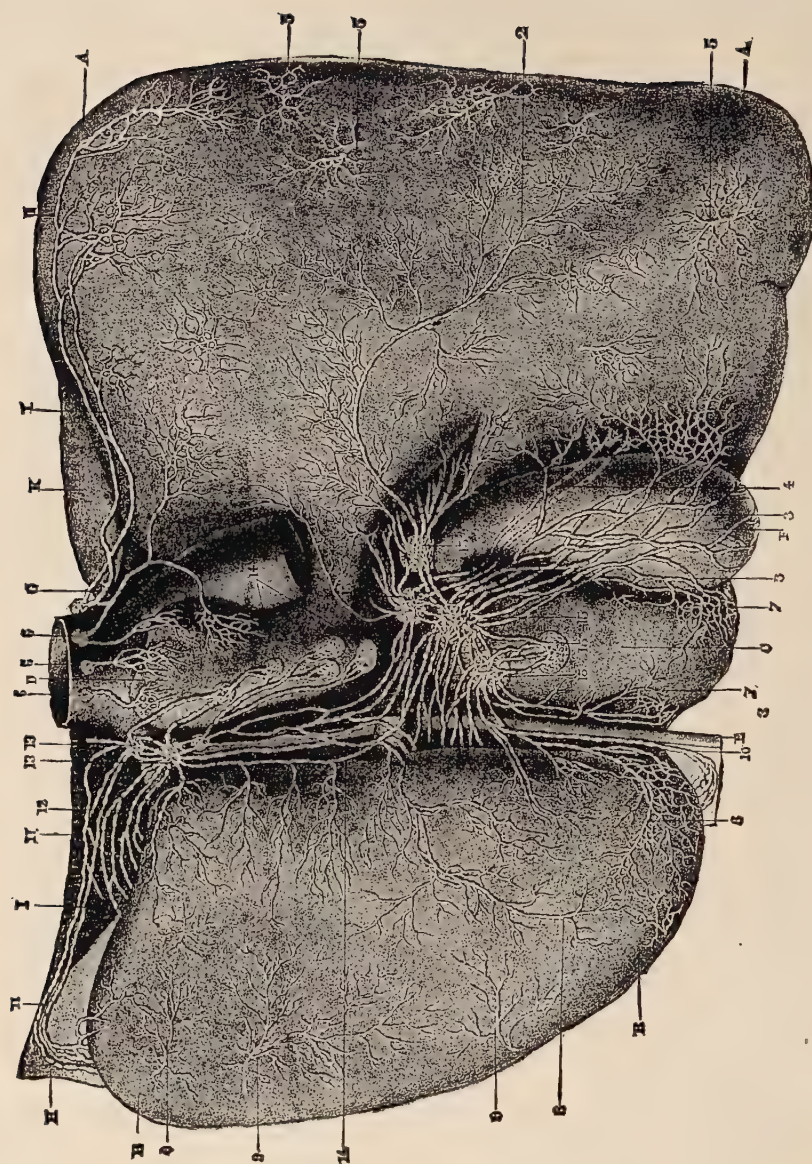


FIG. 6.—LYMPHATIC VESSELS AND NODES OF THE INFERIOR SURFACE OF THE LIVER. (After Sappey.)
 From *Reference Handbook of Medical Science*, Fig. 3869, 1916, Vol. VI, p. 176.
 (Courtesy of Wm. Wood Publishing Co.)

lacteals (lymphatics) from the small intestine pass through the mesenteric nodes, of which there are three tiers, before reaching the large lymphatic intestinal trunk. This trunk and the efferent vessels from the superior aortic (celiac) and mesocolic nodes form one of the most important confluent of the receptaculum chyli.

The stomach lymph is drained through the aortic (upper lumbar) or celiac nodes. Most of the glands are found toward the right extremity, an important fact in connection with carcinoma of this organ.

The vessels of the spleen pass through nodes at the hilus and thence to the celiac glands and to the intestinal lymphatic trunk. Some enter the cisterna directly.

The liver has three distinct sets of vessels. One follows the portal vessels to the hilus, another the hepatic vessels to the post cava group and the third courses through the suspensory ligament to the diaphragm.

The lymphatics of the diaphragm are quite easily demonstrable. One group lies on the inferior surface and communicates freely back and forth with the superior group lying on the upper surface of this structure. Some of the vessels pass into the thorax and again into the abdomen. Groups of glands posteriorly and anteriorly receive the lymph from these vessels. One or two nodes appear near the esophagus.

The lymphatics of the lower portion of the esophagus drain into the vessels and glands of the stomach. Those of the upper portion empty into the cervical (jugular) group, while those in the thoracic portion reach the posterior mediastinal glands. The mediastinal glands receive those vessels from the mammary glands which accompany the perforating vessels, the posterior sternal group, the cardiac and bronchial glands, and in turn drain into both the large lymphatic ducts. Sometimes the vessels of these regions enter directly into either the right or left lymphatic duct.

EMBRYOLOGY

In view of the fact that the structural relationships between the abdominal viscera are not fixed, but are undergoing progressive changes during the latter months of fetal life and for varying periods after birth, it is essential that the student of both the physiological and the pathological problems of these structures should be familiar with their development. Such knowledge will render simple of explanation many conditions which otherwise seem to be very obscure.

Beginning with the primitive trace in the embryo and continuing to full development, there is seen a progressive cell proliferation, a con-

tinuous process of differentiation until the complete separation of the organism into special organs and structures is accomplished. By following out the steps in the process of development one arrives at the explanation of certain embryonal anomalies and vestiges, as the remains of the omphalomesenteric duct, the round ligament of the liver, dilated hypogastric cords, the urachus, Meckel's diverticulum, etc.

Also in this way may be explained the failure of the rotation of the alimentary tract, the reasons for its many convolutions, certain congenital bands and kinks, certain fossæ in the ileocecal and duodenal regions, and also the hepaticoduodenal folds so well described by Nagel.³

By studying the vascularization of the intestine and the short vascular stem, one is able to comprehend how a torsion of a portion (volvulus) or all of the mesentery of the small intestine may occur, also the reason for certain phenomena resulting from thrombosis of the mesenteric veins or those appearing as the result of embolism of the superior or inferior mesenteric artery.

The site of the development of the testis, its relationship to the nephros and to the peritoneal structures, the manner of its descent into the scrotum, the defects in the abdominal wall to permit of this descent and also the relationship of these defects to the development of hernia are much clarified by an extended knowledge of embryology.

The differentiation in the embryo begins by the formation of three folds: the entoderm, the mesoderm, and the ectoderm. From this original cleavage all of the structures are eventually differentiated.

The anterior folds forming from the entoderm coalesce, and the thoracic and abdominal cavities result. The coelom divides into the somatopleure and splanchnopleure.

At about the third or fourth week the splanchnopleure (abdomen) consists of a cavity across which extends a mass of cells from front to back. Within this cellular septum lies the enteric tube. This septum is composed of mesenchymal cells. On either side of this group of cells lies a cavity (the pleuroperitoneal cavity). Each of these cavities is lined with mesenchymal cells, which make up the future endothelium.

Very early disappearance of the cells of the septum in front of the enteric tube below the level of the future stomach takes place. That portion of the septum lying over the stomach becomes peritoneal and forms the lesser omentum. The septum below disappears and the abdomen becomes converted into a single cavity.

While the mesenchymal septum is in situ, liver buds form from the enteric tube developing the liver, and this organ receives its peritoneal covering and its capsule from these mesenchymal cells.

In the portion of the septum lying behind the gastric segment of the enteric tube, the spleen and pancreas appear.

About this time (fifth week) the stomach appears as a bulging of the entoderm and begins to assume its subsequent form. The stomach enlarges and the septum carries within it the gut and from it the mesentery is developed.

By this time the celiac and mesenteric vessels for the nourishment of the stomach, the liver, the spleen, the pancreas, and the intestine are apparent.

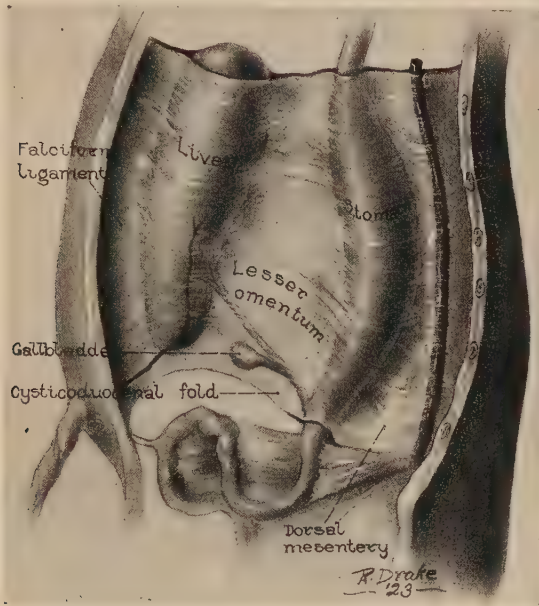


FIG. 7.—DEVELOPMENT OF STOMACH AND LIVER. (After Nagel.)

The stomach soon assumes its more transverse position. The future duodenum, which forms from the midgut and lies somewhat to the right of the midline, becomes fixed to the posterior wall of the abdomen at the duodenojejunal fold, while according to Toldt the duodenum is free with a mesentery continuous with the mesogastrum.

At about the fourth week the foregut, the midgut, and the hind-gut are to be distinguished.

In the foregut can be made out the pharynx with its diverticula, the esophagus, and the stomach.

From the midgut are formed the small intestine and a portion of the large intestine and from it have budded the diverticula giving origin

to the liver and the pancreas. The hind-gut supplies the development of the remainder of the large intestine and the rectum.

At a point somewhat inferior to the pylorus, a budding of the gut occurs which becomes the future cecum, and lies over that portion of the midgut adjoining the right extremity of the stomach. This is the point at which the colon secures its first attachment.

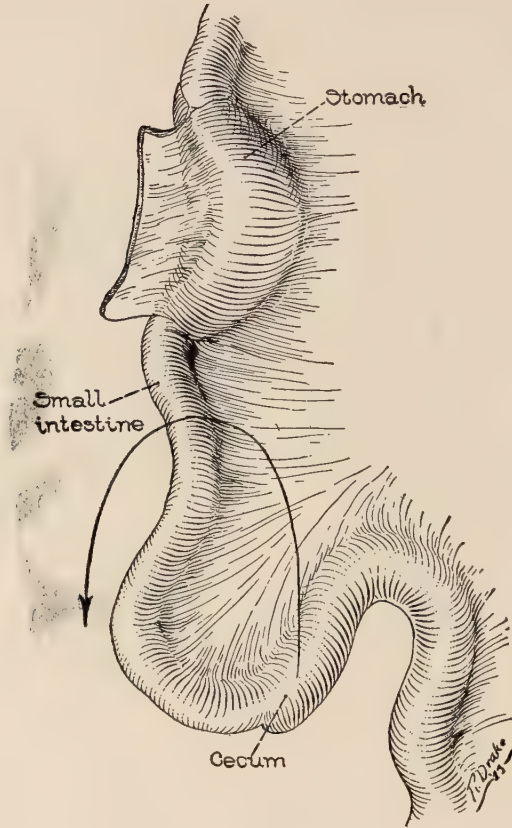


FIG. 8.—FIRST STAGE OF INTESTINAL ROTATION.

During this period the primal mesentery becomes twisted upon itself. As the gut grows it elongates, and this produces the convolutions because the distal portion of the mesentery is so much longer than the proximal part. With the enlargement of the stomach and the development of the spleen and pancreas, the primitive septum (future peritoneum) is carried to the left with the greater curvature. The pancreas also extends to the left and lies behind the future peritoneum. The spleen lies further to the left and retains the peritoneum as a complete covering.

This primitive peritoneum (mesenchymal septum) seems to exceed the apparent needs and begins to pouch to the left and below, forming the great omentum¹.

The great omentum is formed from that portion of the septum which lies below the stomach. The peritoneal coverings on the anterior and posterior surface of the stomach extend downward in contact with

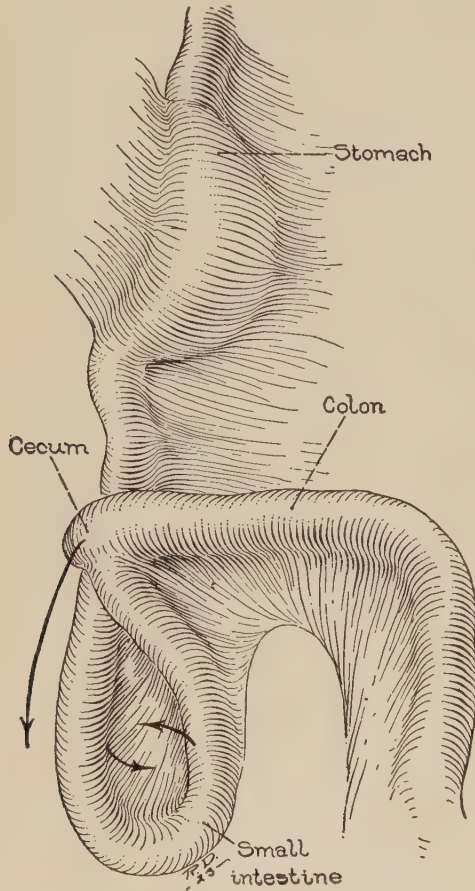


FIG. 9.—SECOND STAGE OF INTESTINAL ROTATION. (After Nagel.)

each other, forming a part of the anterior wall of the earliest lesser peritoneal sac. Reaching to and surrounding the gut at the site of the transverse colon, the two layers then extend upward and backward to separate at the site of the pancreas. One of these layers continues upward on the posterior abdominal wall to form the posterior wall of the lesser peritoneal sac. The other one descends and becomes continuous with the posterior parietal peritoneum overlying the kidney.

After birth fusion takes place between the four layers of the gastrocolic omentum, and there remain only two layers in the great omentum. Occasionally this fusion does not take place and the four layers remain. The space between them communicates with the lesser peritoneal cavity. Retroperitoneal accumulations reach the surface sometimes through the great omentum. I have seen such an occurrence following a chronic

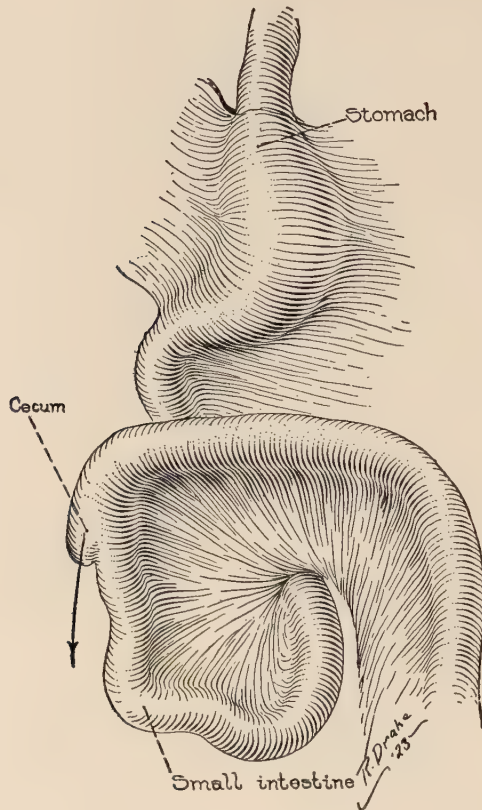


FIG. 10.—THIRD STAGE OF INTESTINAL ROTATION. (After Nagel.)

gastric ulcer with slow perforation and abscess of the lesser peritoneum.

Pancreatic cysts, too, sometimes push forward through this space, sometimes above the stomach and sometimes below the colon.

The question arises whether an organ which has had a mesentery and subsequently lost it, becoming extraperitoneal, did so because of a separation of the two layers of which the mesentery was composed, permitting them to spread out and allowing the developing organ to form attachments to the wall; or whether the organ in its growth through a

fusion of one of its layers with the mesentery of the wall, as claimed by Toldt, produced this result.

It seems quite likely that advancing growth in an organ might cause a lifting of the covering mesentery and a gradual separation of the mesenteric folds at its base, so that finally it becomes completely extra-peritoneal.

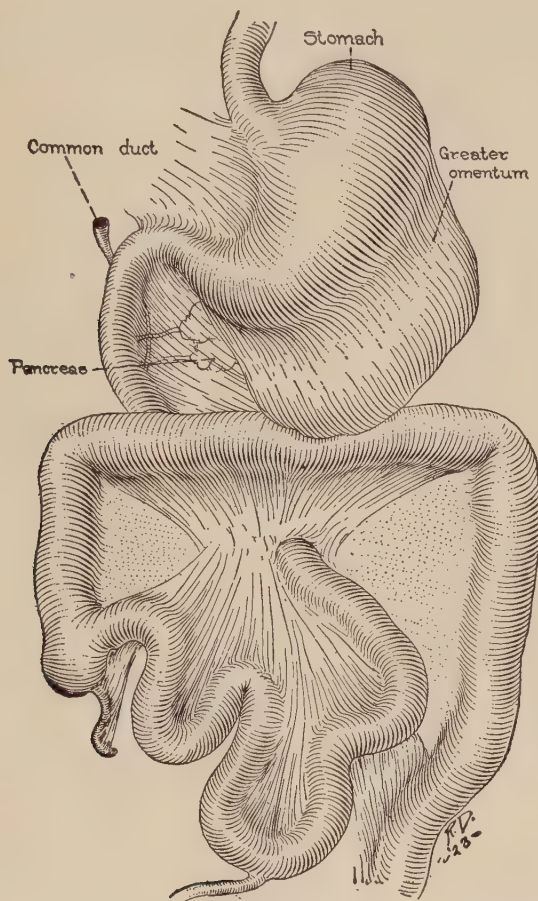


FIG. II.—INTESTINAL ROTATION COMPLETED. (After Nagel.)

In the case of the pancreas this conclusion seems quite reasonable. Again, in the development of the duodenum which remains covered with peritoneum in its first portion where it is continuous with the stomach, there is an abrupt termination of the peritoneal fold which fails to cover the duodenum posteriorly, and does so only partially on either side; while the third portion is almost entirely postperitoneal, the gut again

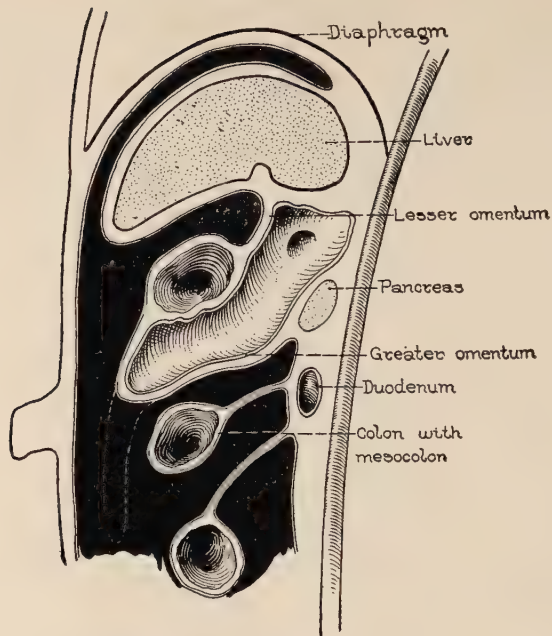


FIG. 12.—THE FORMATION OF THE GREATER OMENTUM. (After Nagel.)

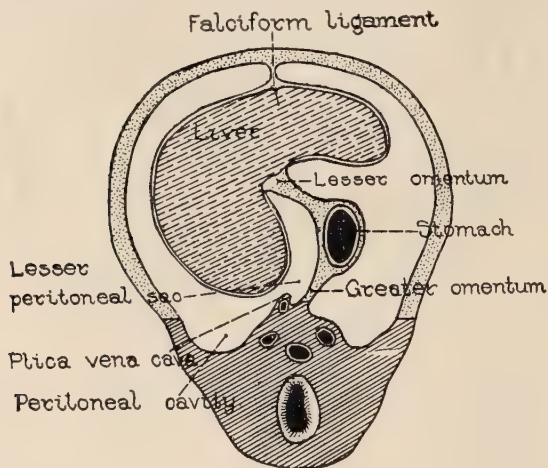


FIG. 13.—THE FORMATION OF THE LESSER PERITONEAL SAC. (After Nagel.)

assuming its mesenteric relation as the jejunum leaves the ligament of Treitz. It seems much more reasonable to conclude that this peculiar arrangement occurred by pushing the folds apart to permit the vascularization of these structures, than to admit Toldt's contention that there occurred a plication of the peritoneum, and that one fold was lost.

In sliding herniæ of the cecum, which develop largely because of a free motility and wide peritoneal covering of the caput coli with an unusual length of mesentery, the act of sliding down into the inguinal canal and scrotum, divests the caput of part of the mesentery, so that a large portion of the cecum becomes firmly attached to the fascia and forms a portion of the sac, thus causing the hernia to become irreducible.

In other instances, where the peritoneal coverings lie in juxtaposition to the peritoneum of the sac, no matter how great the amount of adhesion,

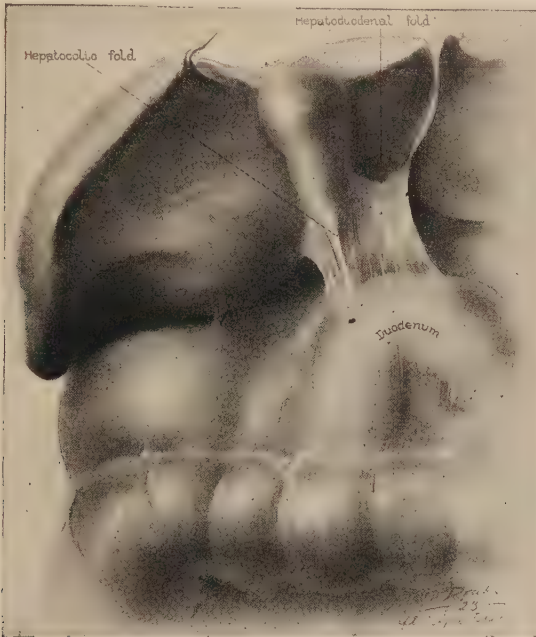


FIG. 14.—LIVER, GALL-BLADDER, FORAMEN OF WINSLOW, GASTROHEPATIC OMENTUM OPENING INTO BURSA OMENTALIS. (After Nagel.)

the herniæ never become extraperitoneal. In the umbilical and ventral herniæ, too, there may be rupture of the peritoneum when the intestines escape, but there is always a serous (peritoneal) covering forming the sac. It is our belief, therefore, that the peritoneum tends to proliferation and rarely to atrophy.

The bladder, as it ascends and descends in filling and emptying, affords an example of how the peritoneal relations change by this method of sliding apart under increase in size.

It must therefore be assumed that the extraperitoneal position of any viscus results from a separation of the peritoneal folds as it develops.

Hertzler² calls attention to the point that it is a fallacy "to ignore the possibility that sliding of the peritoneum, as described by the older authors, does not take place at all." He says, "What actually occurs is that those surfaces which have become fused with other surfaces do so at a very early period before the organs assume the position normal to the adult, and having fused may slide for considerable distances. This is best illustrated in the instance of the cecum. This organ becomes fused with the posterior wall when it lies just lateral to the vertebral column over the site of the future pylorus. As the colon develops it forces the cecum to descend over the kidney. In making this descent it slides partly over the (anlage of) peritoneum and partly carries this structure with it."

We agree that the occurrence is as Hertzler states, but do not see the reason for attributing these results to fusion, when the explanation of progressive growth is all that is necessary, the size of the colon increasing more rapidly than does the peritoneum. This seems again to prove the contention of the older authors. While we are prepared to admit the possibility of fusion of two mesenteric surfaces, yet we claim it is not necessary to accept the rather complicated as against the simpler explanation.

The omentum reaches on the left side from the stomach to the diaphragm and extends down to the colon, forming the phrenocolic ligament. It would seem, from the firmness of attachment and strength of this phrenocolic ligament, that it must contain considerable strong connective tissue. On the right, this peritoneal fold covering a portion of the duodenum is continuous with the peritoneum which extends upward along the vessels to the liver. It also covers the bile ducts and forms the right border of the lesser omentum and the anterior boundary of the foramen of Winslow.

In passing over the cystic duct as it leaves the lesser omentum a process of this peritoneum continues over the duct and a portion of the gall-bladder. As the liver grows, its peritoneal covering, as well as that surrounding the transverse colon, is carried to the right, and more or less fixation occurs at this point, giving rise to the hepatic flexure of the colon. This angulation probably occurs because of the growth of the cecum downward. There sometimes persists in this region a delicate layer of peritoneum which is known as Jackson's veil or membrane. For a description of this membrane the reader is referred to the Coffey monograph (one of this series).

The peritoneal attachments about the duodenum vary considerably and materially affect its mobility. In adults the fixation of the second

portion is quite close. This often necessitates an incision of the peritoneal attachment in order to mobilize the duodenum for access to the lower portion of the common duct, as advised by Mayo.

In the presence of a Jackson veil, if the colon is permitted to fill with feces and become heavy, the tug of its weight may angulate the gut and produce fecal stasis.

There are certain peculiarities in the relationship between the peritoneum and the intestines which may permit the incarceration of a loop of bowel and produce strangulation. These are found about the duodenojejunal angle, in the right iliac fossa and at the attachment of the sigmoid. It is well to bear these defects in mind when symptoms of obstruction are present.

Bursa omentalis.—The lesser peritoneal sac is that portion of the peritoneal cavity which is separated from the greater sac by the torsion of the original mesentery, occurring in the process of development.

The lesser omentum carrying within its structure the common bile duct, the portal vein, the hepatic artery, the nerves and lymphatics to the liver, is quite short, extending only from the stomach to the liver.

The posterior layer of this omentum passes backward from the duodenum to extend up the posterior abdominal wall (the posterior parietal peritoneum) for a short distance and over a portion of the liver to join again the upper end of the posterior layer.

A narrow rounded opening is left, lined throughout with peritoneum, permitting free communication between the lesser peritoneal sac (*bursa omentalis*) and the greater. This opening, the foramen epiploicum, described by Winslow, is generally recognized as the foramen of Winslow. It is bounded in front by the hepatic artery in the hepatoduodenal ligament, above by the caudate lobe of the liver, behind by the vena cava inferior, below by the superior flexure of the duodenum.

It is particularly important because it permits the fingers of the surgeon or a clamp to grasp the entire blood supply to the liver for the temporary control of hemorrhage from that organ. It also has been the seat of strangulated internal hernia. Thirteen cases are on record, twelve of Jacobson² and one of E. Andrews.¹

In some of these cases a large proportion of the intestine has been found within such a hernia. This protrusion passes into the lesser peritoneal cavity which forms the sac of the hernia. Such herniæ are very grave lesions.

The lesser cavity often receives escaping contents from the perforation of a gastric ulcer upon the posterior surface of the stomach. It may

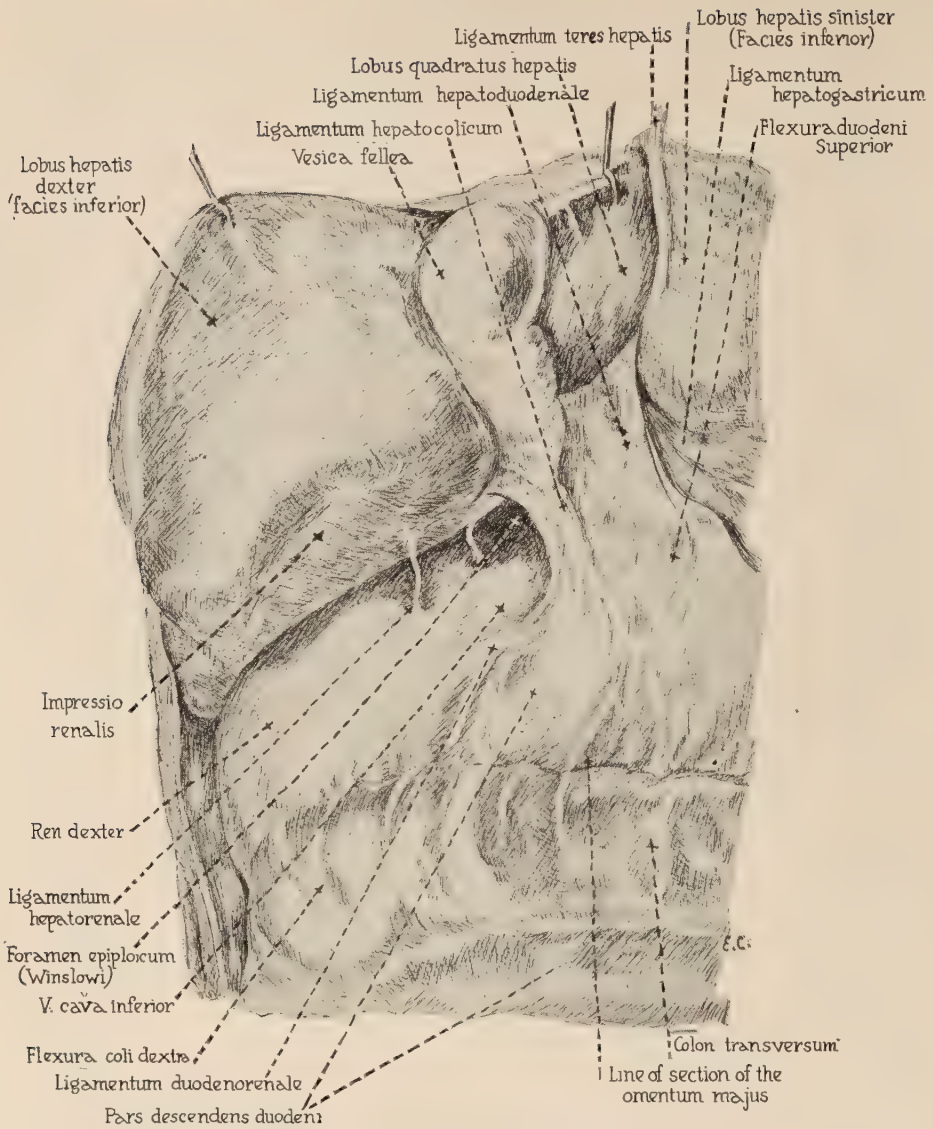


FIG. 15.—FORAMEN EPIPLOICUM. (After Winslow.) (Redrawn from Spalteholz.)

also be filled with bloody fluid or pus from acute or from suppurative pancreatitis.

As a result of either of these conditions an abscess may form in this sac. Such an abscess, because of closure of the foramen of Winslow, may exist for some time and eventually become sterile. It may point in the loin, or it may cause the formation of adhesions between the lesser omentum and the abdominal wall and rupture at the umbilicus.

The omental bursa may be lessened in size in old age by partial fusion of its peritoneal walls.

Treitz has described certain anatomical arrangements about the duodenum which are of sufficient surgical importance to receive consideration.

On the left side of the duodenum, when the transverse colon and the great omentum are lifted, two folds of peritoneum may be seen.

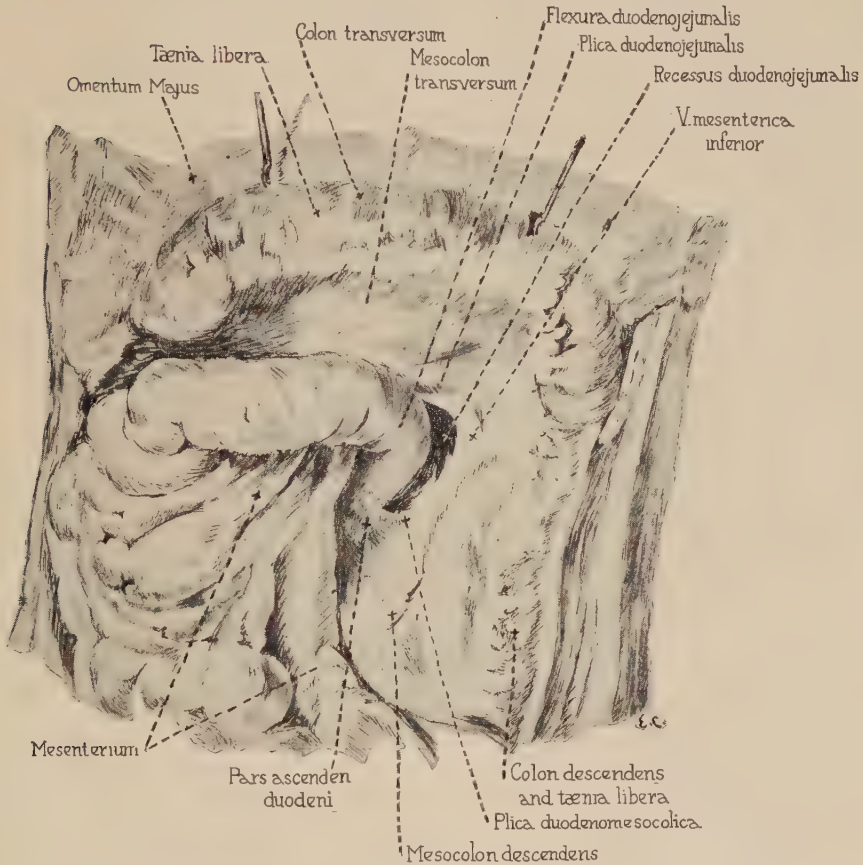


FIG. 16.—DUODENOJEJUNAL FOLDS. LIGAMENT OF TREITZ. SUPERIOR AND INFERIOR DUODENAL FOSSA. (Redrawn from Spalteholz.)

The upper one of these, the duodenojejunal band or ligament of Treitz, extends to the left from the duodenojejunal flexure. It is triangular, or may be considered sickle-shaped with the concavity of the arch downward, and carries within its folds the inferior mesenteric vein. This band marks the upper border of a fossa (superior duodenal) which extends upward and to the left toward the pancreas. Hernia of the

intestine occurs into this fossa (left duodenal hernia). The direction of such hernia is upward and to the left. The mouth of the sac under the ligament of Treitz looks downward and to the right. Sixty such cases are recorded.

Below the ligament of Treitz is another fold, less well developed, is the duodenomesocolic fold, extending from the ascending portion of the duodenum to the left where it is continuous with the mesocolon. This fold has a concavity upward and, with the ligament above, makes an almost circular opening which bounds the recess of the duodenum. The upper portion of this recess is the superior duodenal fossa, and the one below is the inferior duodenal fossa. Hernia into this fossa extends downwards and to the right, with the opening upward and to the left.

Occasionally these folds are obscured by a prolongation of the mesentery downward from the duodenojejunal flexure for several inches.

On the right of the ascending duodenum there appears a fold of mesentery in which lies the superior mesenteric artery as it courses downward from its origin from the aorta and over the pancreas. This outlines the mesentericoparietal fossa of Waldeyer. This fossa may form the sac of a hernia (right duodenal hernia). Seventeen cases are recorded.²

Ileocolic Fold.—The anatomical arrangement about the caput coli varies very greatly in different individuals. This arrangement depends in a great degree upon the stage of development at which the observation is made. The anomalies occurring in this region are quite numerous.

There are, however, certain folds usually observed in this region which are of sufficient surgical importance to be mentioned. The different names applied by different writers are somewhat confusing and an effort will be made to employ only those most in use.

Where the ileum joins the cecum the usual arrangement of the peritoneum shows a reduplication. The ileum is covered anteriorly for a short distance by a fold of peritoneum, the superior ileocolic fold of Waldeyer (anterior vascular fold of Moynihan). This fold extends from the mesentery of the ileum and passes over the small intestine to be attached to the colon. Sometimes it extends as far down as the base of the appendix. It lies over the ileocolic artery and sometimes carries a branch corresponding to the appendicular, coming off with the latter from the ileocolic artery. When the appendicular artery is large, the one in the ileocolic fold is small. Sometimes in such event the fold is small or absent.

The appendicular artery traverses the fold of mesentery known as

the meso-appendix or posterior vascular fold, which lies posterior to the ileum.

The superior ileocolic fold of Waldeyer (anterior vascular fold, Moynihan) forms, with the ileum and above this part of the intestine with its mesentery, a fossa extending to the right border of the colon, the ileocecal fossa, which is sometimes the site of a hernial protrusion. This form of hernia pushes upward along the colon and over the kidney region.

In front of the meso-appendix there sometimes passes a fold, the bloodless fold of Treves, or the superior ileocecal fold of Waldeyer. This is a quadrilateral band extending from the ileum to the cecum just anterior to the meso-appendix. Between this fold and the meso-appendix a hernia may form, the intestine passing backward between the ileum and the cecum, but anterior to the meso-appendix: the ileo-appendicular hernia.

Another fossa lies behind the meso-appendix, bounded externally by the cecum and internally by the ileum: the inferior ileocecal fossa, within which the appendix sometimes lies entirely submerged. Hernia may also occur in this space. The cecal fossa lies behind the cecum, which must be raised to bring it into view. It varies widely in size, and extends upward behind the ascending colon toward the kidney.

The intersigmoid fossa is situated at the base of attachment of the sigmoid to the pelvic wall at the ilio-sacral junction on the left side.

Folds and Fossæ of the Lower Abdomen.—In the lower portion of the abdomen are found certain cords and along with them certain fossæ (see Fig. 1). These cords and fossæ are of some importance in relation to the occurrence of hernia.

An observation of the anterior abdominal wall from the peritoneal side shows in its lower portion a number of cords extending upward in a general direction toward the umbilicus. The central of these cords is the urachus, which extends from the fundus of the bladder to the umbilicus. It lies under the peritoneal surface and is sufficiently prominent to be seen readily. On each side extending from the pelvic brim along the abdominal wall and approaching each other and the urachus as they ascend are the remains of the hypogastric cords. Still farther out and running less obliquely upward lie the deep epigastric arteries.

Because of the elevation of the peritoneum by these structures and from the prominence of Poupart's ligament there may be observed four fossæ on each side. One, the supravescical fossa, lies between the urachus and the hypogastric cord on each side. On either side of the deep epigastric artery lies a fossa, the mesial and the lateral inguinal fossa. Below Poupart's ligament and internal to the deep epigastric artery is the

femoral fossa through which protrusion of femoral hernia occurs. Indirect or external inguinal hernia emerges through the lateral inguinal fossa. Direct inguinal hernia occurs through the median fossa.

Mesocolic Fold.—On the left side is occasionally found an opening in the mesocolon, through which the intestine may pass. Only two cases have been recorded, those of Dodson and the earlier one of Sir Astley Cooper.

ANOMALIES

Meckel's Diverticulum.—This is one of the most frequent congenital anomalies found in connection with the peritoneal cavity. It is a portion of the omphalomesenteric duct, an embryonal structure connecting the gut with the allantois sac which persists in some individuals, in part or throughout its entire extent, after birth. It is usually about three inches in length, connected by one extremity with the lower portion of the ileum. The other end is free or may be continuous as a cord to the umbilicus.

The structure of the diverticulum is identical with that of the gut of which it is really a part. Its lumen is continuous with that of the intestine. In very rare instances it remains patent and persists as an omphalomesenteric fistula at the navel. In some cases the external portion of this structure persists as a red, strawberrylike mass protruding at the umbilicus. The mucous membrane persists and is continuous with the skin, but no communication into the diverticulum is present. This is called an omphalomesenteric diverticulum.

Instances in which the structure closes at each extremity, forming a cyst in the remains of the omphalomesenteric duct, are extremely rare.

Clinically Meckel's diverticulum is particularly important as a causative factor in internal strangulation of the intestine. This may occur from a loop of intestine passing around a diverticulum in which the attachment to the parietal wall has persisted, or it may occur as the result of an acquired attachment making a band of the diverticulum. Such bands have been observed pressing upon a neighboring loop of intestine and either partially or completely occluding it as would a string or tape. It also favors the occurrence of volvulus by torsion of the intestine around such a band as a pivot.

Inflammation, diverticulitis, sometimes develops and closely resembles peritonitis.

The presence of Meckel's diverticulum may be made out through a skiagram even in the absence of symptoms. Whenever the symptoms

of internal strangulation occur, this must be looked upon as a possible cause.

There is considerable variation in the point of attachment to the intestine according to G. S. Huntington, who made studies upon this point in 22 cases. The distance from the cecum averaged 107 centimeters. In diameter it is about one third that of the small bowel. It may be recognized from the appendix by the fact that it springs from the ileum, while the appendix arises from the cecum at the extremity of the longitudinal muscular bands. Usually the end of the diverticulum is rounded and smooth, but may continue as a cord attached to the abdominal wall. It is larger as a rule than the appendix.

Many cases of Meckel's diverticulum occur in which no symptoms are present to attract attention to it. When inflammation is present sufficient to give rise to considerable discomfort and when this rudimentary organ acts as a causative factor in ileus, or when peritonitis is excited as the result of either of the above conditions, operative intervention is indicated.

When internal strangulation or volvulus is present, prompt abdominal section is indicated, as well as when the symptoms are less acute, but persistent and annoying.

Transposition of the Viscera.—Transposition of the abdominal organs occurs in total transposition of the viscera. Partial situs transversus is much less frequent than total transposition which is itself quite rare.

Its particular importance clinically lies in the fact that failure to recognize it may result in errors in diagnosis. Recognition of this arrangement occurred very early in medical literature. Aristotle's writings show that he had observed two cases in which the organs were transposed in animals. Fabricius³⁰ in 1600 related a case of reversed liver and spleen. The first recognized instance of its total occurrence in man is attributed to Petrius Servius in 1643.⁵ Carl Beck⁷ states that the first authentic case was reported in the time of Molière, in the person of Marie de Medici, Queen of France.

Frochel, in 1745,³⁰ made a brief citation of partial inversion of abdominal viscera in the course of a wider discussion of general malformations of the viscera. Cornelius Gemma,³⁰ 1769, was the first to make special study of such cases. Küchenmeister³⁰ first recognized the condition during life by methods of physical examination.

In Grueber's⁶ 79 cases collected from the literature up to 1865, only 5 were recognized during life. Küchenmeister⁶ collected 149 cases up to 1888, which number was increased by Pic⁶ to 190, in 1895.

Karashima³⁰ in 1912 reviewed over 200 cases and gave what is perhaps the best historical résumé.

Arneill⁶ states that partial situs inversus occurs less frequently than complete transposition. "Lochte, up to the year 1894, collected thirteen cases of this incomplete variety. In more than half the descriptions were very incomplete." Arneill concludes that a total of 300 cases had been reported up to the time of his communication, 1902. He reports 4 cases coming under his own observation from 1897 to 1902, and mentions 37 cases collected from leading practitioners. Of these all except 6 were discovered during life.

It appears from these reports that cases of visceral transposition are recognized with relatively much greater frequency during life than was formerly the case.

Sherk³⁹ in 1922 reported a case of his own and 24 cases recorded since 1912, also 3 verbal reports from J. M. King, S. J. Mattison, and Hubert A. Royster, but stated that these figures could not be considered as accurate or complete, since this anomaly is reported under different titles and individual cases are often duplicated. He mentioned a hurried search from the Mayo Clinic which shows 10 cases indexed since 1910. For the same period their registration was 347,000. He also stated that only two writers, Lane²⁶ and Parsons-Smith,³⁵ mention observing this condition in recruits during the late war.

This abnormal visceral arrangement was found 49 times in women and 19 times in men in Grueber's series,⁶ while no mention of sex is made in 11. "These individuals lived as long as those with normally placed organs. The women were normally fruitful. Only four of the group were extremely malformed." There was transposition of both the thoracic and abdominal organs in 71 and of the abdominal alone in 8 cases.

In this form of situs partialis the abdominal organs may be transposed in a very irregular manner. In fact it is likely that transpositions of the partial type are in many cases due to failure of rotation. Strictly speaking such a type is quite different in origin from total visceral transposition. The determining factors in total transposition are not clear. In fact there has been no certain explanation of the normal left-sided development of the heart or explanation why the usual arrangement of the abdominal viscera preponderates over the rarer occurrence in which the liver and the other abdominal organs are reversed. It appears logical to conclude that the usual situs is determined by certain unknown developmental factors. Von Baer⁶ considers that the embryo normally lies on the left side of the umbilical vesicle and that this position determines the left-sided position of the cardiac apparatus and the usual arrangement

of the organs. According to his view, those cases in which the embryo lies on the right side show a transposition of the organs. His theory yet leaves for explanation the reason for the marked frequency of one type as against the infrequency of the other type of development. According to von Baer these changes take place at the beginning of the developmental period.

Forster ⁶ also considers the situs inversus as a malformation in which the transposition of the anlagen takes place in the first embryonal formation.

In double monster the fetus of the right side shows a complete transposition, while the fetus of the left side shows a normal situs.

Rindfleisch ⁶ thinks the spiral turning of the blood column is responsible for the displacement of the heart. The normal flow is from left to right, while in situs inversus an opposite direction obtains. The asymmetry of the heart is considered responsible for all the asymmetry in the animal body.

Virchow ⁶ emphasizes the importance of the umbilical cord. In situs inversus it is wound spirally to the right while normally it is to the left. This seems to the author to be of little importance since the twist in the umbilical cord probably is determined by intra-uterine movements of the fetus.

Küchenmeister ⁶ considers the location of the fertilized disk at the surface of the egg is the essential thing. The normal situs in single birth probably depends upon the growth of the germ from below upward instead of from above downward. He says that "from this it must be self-evident that the turning of the embryo has been inverted. This must also affect the later spleen side and the side of the arterial heart." Concerning the congenital partial situs viscerum—solito inversus—which shows itself either in the chest or belly, but not in both places at the same time, he believes that "the growth, on the whole, follows the type for the situs inversus. The rarer partial situs is an inhibition formation which grows according to the type of the normally projected embryo."

It must be admitted that the various explanations for transposition of the viscera are only theories and none of them shows sufficient proof to receive general acceptance. Clinically such cases assume importance since the superficial observer may very easily overlook the possibility of this occurrence and considerable error in diagnosis result.

Virchow ⁷ records a case in point where, notwithstanding the observation of reversed ordinary physical signs, the clinician had stated that on auscultation the heart sounds were "heard with difficulty on account of the presence of bronchitis," and that the "spleen was very much en-

larged." In this case the heart murmur had not been looked for upon the right and the liver had been mistaken for the spleen.

It can be readily understood how visceral transposition could be overlooked formerly when the entire dependence of the observer was necessarily placed upon methods of physical diagnosis. Moreover the infrequency of the condition resulted in little consideration being given to the possibility of its occurrence. More attention has been attracted to this subject lately, and this fact coupled with the improvement from roentgenoscopic methods reduces the possibility of error very greatly. This is clearly shown in the greatly increased accuracy of diagnosis in recent reports.

Beck⁷ reports an interesting case of gall-stones developing in a transposed liver which presented some difficulties in diagnosis. The clinical recognition of the presence of situs viscerum transversus becomes simplified when attention has been called to it. The clinician, therefore, should not overlook the possibility of its occurrence, particularly because of its infrequency.

The comparative frequency with which transposition of the thoracic viscera accompanies that of the abdominal organs makes discovery of the latter condition by a careful observer more probable. The displacement of the heart to the right, particularly with reversal of the position of the apex beat, the abnormal position of maximum intensity of the heart sounds, the transposed area of dulness, make recognition of dextrocardia certain in moderately thin individuals. Fluoroscopic examination quickly confirms the physical findings even in fleshy persons in whom occasionally it may be difficult to accurately outline the heart. The presence of dextrocardia should at once attract the attention to the possibility of transposition of the abdominal viscera. The absence of liver dulness upon the right and the presence of a corresponding area of dulness upon the left side together with gastric tympany in the right hypochondrium point strongly to this anomaly.

Failure to recognize hepatic dulness in the right hypochondrium in normally placed livers must be borne in mind. When such a finding is observed in the presence of a splenomegaly or a renal tumor upon the left side the diagnostic acumen of the observer may be considerably taxed. The sharp margin of the reversed liver resembles that of the spleen so closely that it may be difficult by physical examination alone to make a differentiation. All types of splenic enlargement show marked changes in the blood picture which becomes a material aid in diagnosis. The enlargement of a right-sided spleen may be easily mistaken for a normally placed liver in a case of transposition.

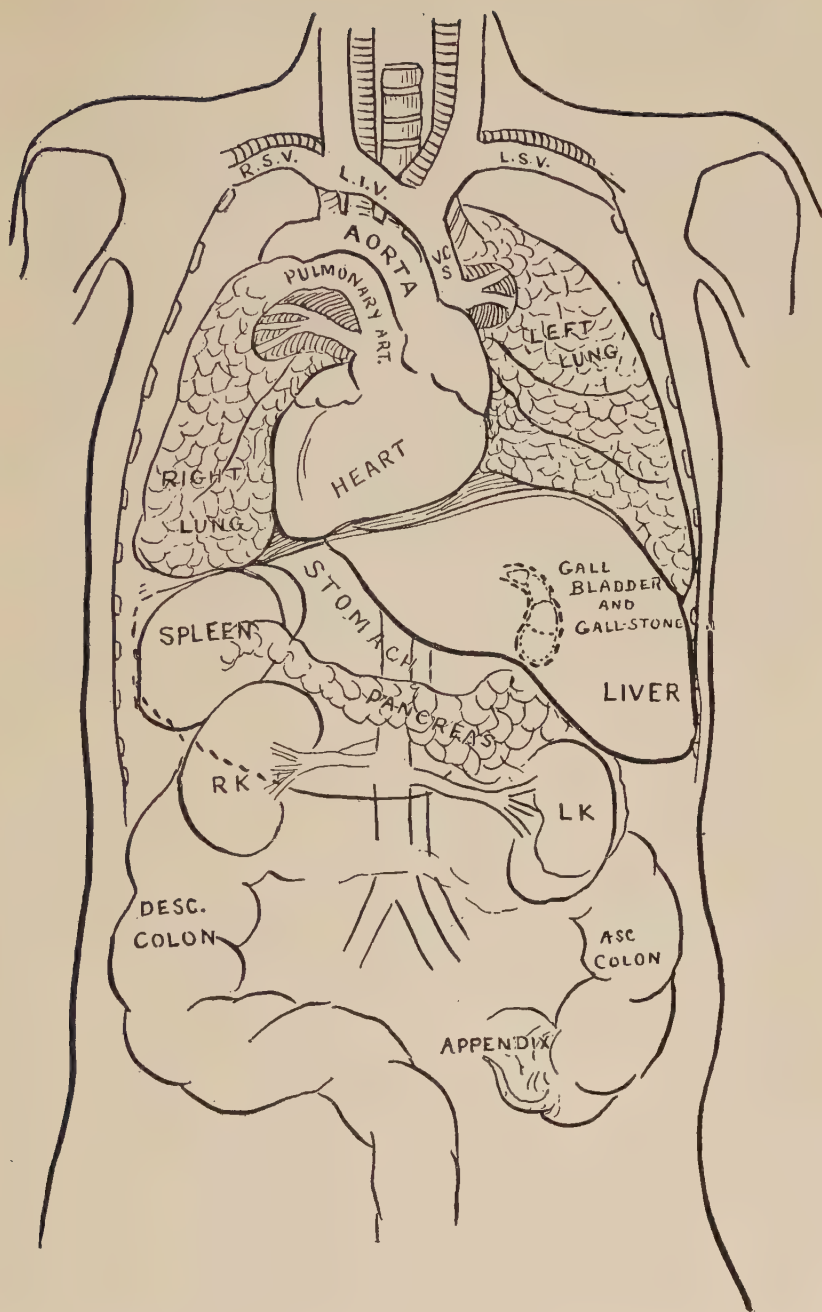


FIG. 17.—TRANSPOSITION. (From Beck, *Annals of Surgery*, Fig. 4, 1899, 29: 599.)

The value of a gastroenterologic x-ray study in the diagnosis of visceral transposition can scarcely be overstated. In cases of doubt after employment of the usual measures pneumoperitoneum may be added to the technic to determine the exact visceral locations.

As previously stated, in the majority of cases of transposition of the viscera there is no interference with usual function or with the longevity of the individual. These cases are important largely from their infrequency and because of the necessity of considering this occurrence when attempting the diagnosis of intra-abdominal lesions.

Failure of Rotation.—There are also certain departures from the usual in the anatomical relations in certain regions which properly may not be called abnormalities. They are, however, sufficiently infrequent to be included under the head of anomalies.

Such an anomalous arrangement occurs in the right upper quadrant. Certain bands or folds of peritoneum are sometimes seen in the region of the gall-bladder at operation or necropsy. A considerable proportion of these structures may be demonstrated as inflammatory in origin. Others are undoubtedly of congenital formation.

The most recent contribution to the study of these structures in the neighborhood of the gall-bladder is that of Gunther W. Nagel,³ to whom and to the Mayo Clinic I am indebted for permission to use his plates. He makes particular mention of folds extending from the fundus of the gall-bladder to the duodenum and the transverse colon.

Most standard works on anatomy either fail to mention these folds or dismiss them lightly. A few, as Piersol, Testut, and Belou, describe a ligament extending from the gall-bladder to the upper border of the transverse colon.

Nagel states that Virchow specifically calls attention to the frequent attachment of the right colic flexure to the under surface of the liver and gall-bladder, and also of anomalous bands from the latter to the pylorus and duodenum, evidently referring them to a chronic localized peritonitis.

Nagel considers the folds which he describes as cysticoduodenocolic ligaments to be prolongations of the lesser omentum to the under surface of the gall-bladder, which extend to the right across the duodenum and downward toward the transverse colon where they become continuous with the anterior layer of the greater omentum. His conclusions are:

1. A cysticoduodenocolic ligament is present in a small percentage of persons.
2. The ligament is a part of the lesser omentum and therefore of congenital origin.

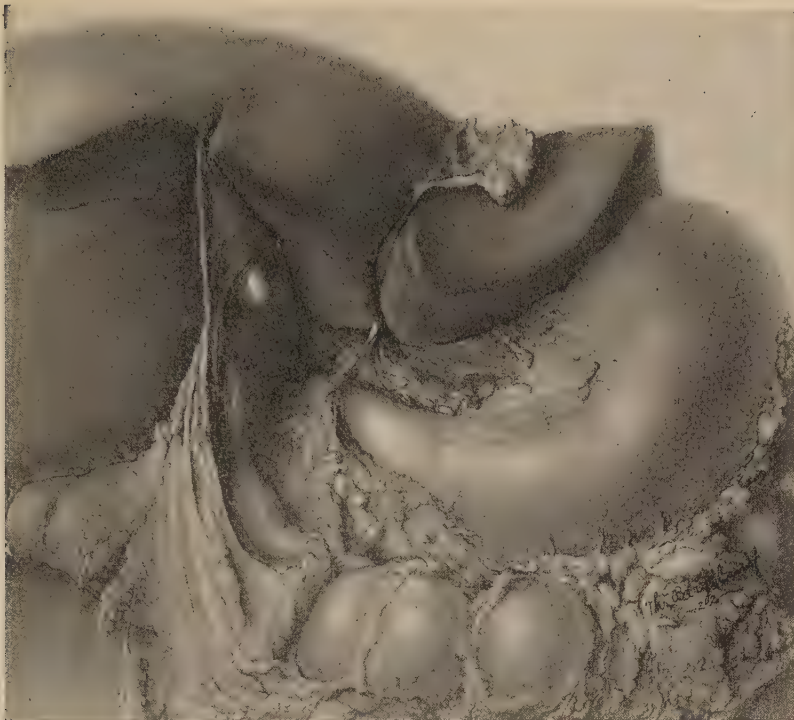


FIG. 18.—WELL MARKED CYSTICODUODENOCOLIC LIGAMENT. (After Nagel.)

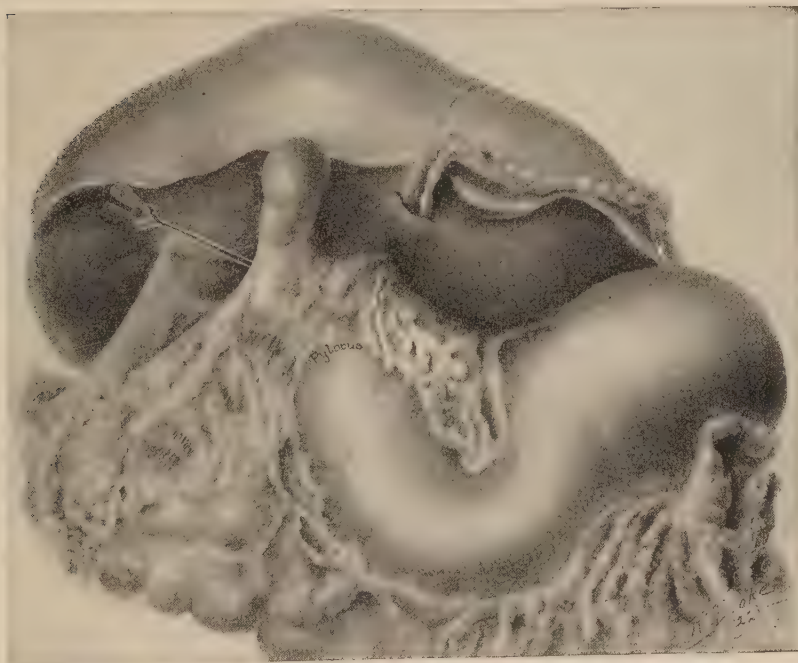


FIG. 19.—EXTENSION OF THE ANTERIOR FOLD OF THE LESSER OMENTUM. (After Nagel.)



FIG. 20.—ILLUSTRATING ANOMALOUS ARRANGEMENT OF THE CECUM AND APPENDIX
SHOWING AN OBLIQUE INSTEAD OF A TRANSVERSE COLON.

Note the proximity of the appendix to the gall-bladder. It would be easy in such a case to mistake inflammation of one for the other. Incomplete rotation. Entire colon on right side mobile with a two-inch mesentery.

3. Under normal conditions the ligament probably produces no symptoms and therefore requires no treatment.

4. The ligament is, however, a source of danger in that it represents a potential adhesion ready to thicken and shorten in response to neighboring pathological processes, and to furnish a guide and stimulus to the formation of true adhesions.

One very interesting case of anomalous arrangement presented in a man thirty years old. The cecum appeared as a funnel shaped pouch entirely covered with peritoneum and having a short mesentery. It was continuous obliquely across the abdomen with the transverse colon to the splenic flexure, this entire portion of the intestine being quite mobile. The caput had not descended to its usual location but lay just below the liver and above the sacro-iliac articulation. The appendix, which was eight inches long, was continuous with its lower extremity. It curved to the right and extended around the right margin of the colon, lying external, above, and somewhat behind the colon. The tip was curled on itself and lay below and in contact with the gall-bladder. A peritoneal process, continuous from a rather short broad omentum, extended to the right side covering the colon and the coiled appendix and was attached to the parietal wall. This layer seemed to be a thickening of the mesocolon. At the proximal extremity of the colon it was about two inches in length and continued to the left at the pelvic brim covering the terminal portion of the ileum. The latter approached the point of junction with the colon in a semicircular curve from the sacral region concavity upward. It was held firmly in place by this peritoneal fold and its posterior surface was closely attached to the posterior parietal wall. The vessels for its supply which ordinarily appear in its mesentery could be seen coursing through the posterior parietal peritoneum at the pelvic brim.

This case apparently confirms the contention of Lane and Jackson that such arrangements as they have described are congenital in origin. There were no evidences in this case of any signs of an inflammatory process, either in the appendix, colon, or ileum. This specimen was obtained from a man thirty years old who had died from pericarditis following a severe wound that involved the pericardium, the heart, and the right lung.

It seems more rational to explain the conformation in this case by an arrest of development rather than as the result of inflammatory changes. This opinion seems confirmed by the failure of fixation of the colon to the right parietes. There was no hepatic flexure present, the colon passing in a straight direction obliquely across the abdomen from

the right lower quadrant to the splenic flexure. The ileum entered the colon from below.

This, with many similar cases, seems to point to the congenital origin of many of the anomalous folds, bands, and kinks described in the literature. Deavers' contention that they are all of inflammatory origin does not seem tenable, even admitting the possibility of prenatal inflammation. This does not explain all the anomalies. It seems more reasonable to conclude that many of them are due to the persistence of a fetal type from

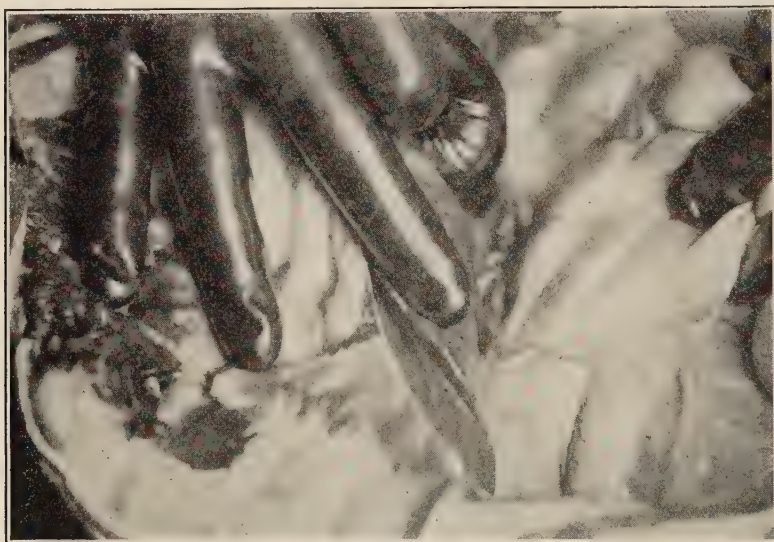


FIG. 21.—ANOMALOUS ARRANGEMENT IN ILEOCECAL REGION.

The ileum has no mesentery. Its posterior surface is closely attached to the pelvic wall. It enters the colon from below. Notice the blood-vessels and lymphatics in the posterior pelvic parietes. Liver in right hand. Appendix shown beneath the liver. Caput coli retracted by left hand. Ileum coursing from right side of pelvis posteriorly.

arrest of development, while some form after birth from inflammatory processes.

Inflammatory bands have a tendency to change in size and extent, both from progression and retrogressive processes, while congenital anomalies have a tendency to persist.

As stated elsewhere, clinically the important thing for consideration is not whether a band, fold, or kink is embryonal or not but whether it is producing interference with the fecal flow or not. The patient is not interested particularly concerning the method of formation of such abnormalities, but whether his health is impaired by its presence. He is also not concerned as to its location, but whether symptoms arising from

it can be relieved. The object, therefore, is to determine the best method of restoring the part to proper function with the least disturbance to the individual.

There is one difference between those veils, bands, and membranes which are the result of failure or arrest of development and those which are formed after birth from inflammatory processes. Congenital arrangements do not in any marked degree change either in extent or relationship and never undergo retrogression, whereas all inflammatory formations show such tendency. They either increase with the persistence of the inflammatory process or diminish with its subsidence. In

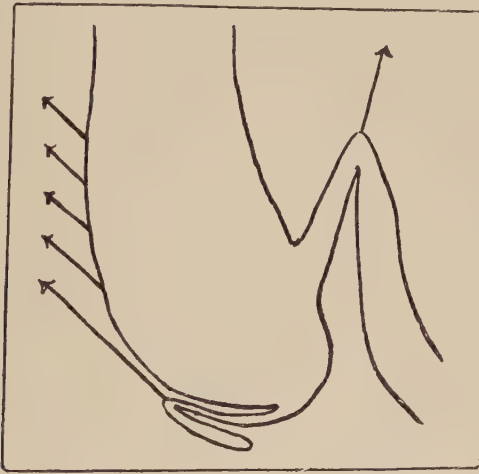


FIG. 22.—ILLUSTRATING THE PULL DOWN AND TO THE LEFT FROM A LOADED CECUM. (After Lane.)

some cases they may disappear entirely, as in Dowd's case. This fact alone may be sufficient to determine, when such bands are found at operation, whether they are inflammatory or not. There will always be present some distinct evidence of an inflammatory reaction, and in many cases lesions in the intestine, stomach, gall-bladder, or other structure will point to its inflammatory nature.

In chronic serositis and in fibrinous tuberculous peritonitis, conditions in which adhesions are prone to occur in profusion, the condition of the diseased intestine will most certainly indicate the reason for their formation.

The congenital anomalies and developmental errors are most often found in certain particular localities where the embryologic process necessitates changes in location and fixation of certain organs. While such

anomalies and aberrant types differ considerably, there are certain salient similarities between them that permit of recognition.

The changes which take place in congenital structures consist in sliding of the peritoneal attachments of the colon, and in sliding hernia resulting from the pull of the intestine as it protrudes into the sac. A similar pull downward and to the left is seen in cases of Jackson's veil or Lane's kink, resisted by a firm attachment to the parietes or one of the viscera. This is well illustrated by the direction of such lines of force.

This pull may be sufficient to produce delay in the transit of the intestinal contents, but it rarely results in actual strangulation. On the other hand inflammatory bands may form in any locality and may soon persist in their formation and subsequent contraction until partial fecal stasis is observed, and complete ileus is not at all infrequent. Cicatricial connective tissue contracts very remarkably, while non-inflammatory congenital peritoneal bands do not contract, but rather have a decided tendency to stretch. Inflammatory bands often become elongated and attenuated, partly from absorption and atrophy from lack of function, and partly from intestinal movements and traction upon them.

Kinks and Bands.—A very close distinction should be made between the membranous folds resulting from embryonal or fetal changes and those forming after birth. The latter are considered under the term abdominal adhesions and their behavior as well as their effect upon intestinal functions differs considerably from prenatal structures.

Inflammatory adhesions in their manner of formation, the changes taking place in their structure and the variety of their action upon intestinal motility, while resembling in part the effects of embryonal peritoneal arrangements, have very much greater pathological importance. This portion of the study will take cognizance of the prenatal structures which form bands, veils, and kinks.

The voluminous amount of literature upon this subject proves that the explanations of the developmental processes taking place in their formation are not easily grasped or capable of positive demonstration.

The theories offered to explain the development of bands, kinks, folds, and veils are many and varied. Of these no one contention seems to explain all the phenomena present in different cases. This is true because the same factors are not active in their production in different cases. Some are certainly embryonal, while others are of fetal inflammatory origin. Adhesions, on the other hand, are always either inflammatory in origin, or as claimed by Lane,²⁹ Fagge, and others, result from the static pull of the overloaded intestine. It would be more correct to state that the traction produced by the weight of an overloaded intestine

causes attenuation of normal peritoneal folds or those arising from inflammatory conditions, thus resulting in kinking of the gut.

Lane ²⁷ considers that bands about the terminal ileum represent the crystallization of lines of force which increase with age and that this effect is the result of efforts upon the part of the organism to support a sagging viscus. This contention, while a most plausible explanation of the probable causative factor in a certain number of instances in which congenital structures are clearly attenuated by such lines of force, seems to be disproved as the chief factor by the observations of John Bryant,¹¹ of Boston.

In discussing the subject of visceral adhesions and bands, normal incidence, before the Gastro-enterological Association, Washington, May 2, 1922, Bryant reports his observations based upon a study of 297 unselected necropsies of all ages and both sexes. This rather exhaustive report divides the cases into two groups, one below and the other above the age of forty. Thus he makes forty years the dividing line between youth and age. In these two groups are included also the fetal and senile subgroups. He says: "Obviously, in the point of total cases, the fetal and senile groups are too small to be more than suggestive; but they are at least suggestive."

He states "that the adhesions found in the fetus are relatively uncomplicated in character and that complexity is practically a synonym for age. In other words, the increasing complexity of adhesions with age seems to be an indication of the decreasing resistance of the viscera to the trauma of all kinds to which they are subjected throughout life.

"In this fetal group the distinguishing characteristics of congenital or developmental adhesions lie in the limited average number of viscera involved as well as in the simplicity and absence of variety in type of the adhesions themselves.

"On the contrary the acquired and degenerative adhesions of age are characterized by the large average number of viscera involved, the great increase in the variety, and the strikingly increased complexity of the adhesions themselves.

"In this series the rate of involvement of the different organs is higher for several organs in the fetus than at later ages, as for the transverse colon in the male and the terminal ileum in the female. This rate increases rapidly with progressive age for certain other organs, as the sigmoid flexure in the male and the adnexa in the female.

"The terminal ileum shows in both sexes a decreasing ratio of involvement by adhesions with progressive age, the rate being distinctly highest in the fetus."

These findings are diametrically opposed to the contention of Lane mentioned above, and appear to disprove the importance of the loaded bowel as directly causative, by its weight, of the formation of these bands. That a loaded intestine by the coincident inflammatory process excited may produce adhesions cannot be denied.

Certain organs on the other hand show a distinct tendency to an increase in the number and extent of adhesions. This is particularly true of the sigmoid, the omentum, and the uterine adnexa.

Certain authors propound the theory that a considerable number of bands presenting at birth are not the result of embryonal errors or of imperfect development, but are really the result of inflammation of the peritoneal surfaces occurring during fetal life.

While it seems that the possibility of fetal inflammatory disease may be productive of bands and the coincident kinking of the intestine, it is unlikely that this is of frequent occurrence. The larger number of non-inflammatory congenital peritoneal folds are embryonal in origin.

The characteristics of structures of this type consist in the delicate nature of the tissue, its smoothness of surface, the regularity of vessels, its lack of tendency to contraction and its persistence.

Such structures form in the course of the development and the rotation of the viscera within the abdomen. Failure of complete rotation and malposition tend to leave certain of the peritoneal structures in an abnormal relation to the intestine, and when these persist they become bands or veils. No complete explanation has been offered for this type of arrested development. All tissues in the process of development show a tendency to an excessive cellular production. That this is also true of the mesenchyma in the development of the peritoneum is apparent. The tendency to absorption of any excessive tissue produced in developmental processes is also shown in these structures.

Because of certain unknown factors the developmental processes lose their impetus and these changes do not go on to completion. In these cases the bands persist as pericolic membranes, etc. Certain of these congenital bands are long and attenuated, while others are quite short. The latter are seen in some instances holding two adjacent folds of intestine in such position as to form a kink. Occasionally, but much more rarely than from inflammatory bands of later life, intestinal stasis results from such bands and kinks.

Patients presenting with stasis of this type usually give a history of sluggish intestinal function and digestive disorder from infancy. In many instances there seems but little change in the amount of discomfort from time to time. In other instances there is apparently a ten-

dency to increased stasis as age progresses. This rarely occurs to the same extent as it does in cases resulting from inflammatory adhesions.

Jabez Jackson,²¹ in December, 1908, before the Western Surgical Association, described a structure of this type which has since been known as Jackson's membrane. This membrane is described as extending from a point just at the hepatic flexure to three inches above the caput coli and from the attachments between these two points continues from the parietal margin over the external lateral surface of the right colon to the internal longitudinal muscular band as a thin vascular veil. In this structure long straight unbranching blood-vessels course, most of which are parallel with each other and "take a slightly spiral direction over the colon from the outer upper peritoneal attachment to the inner lower portion of the gut, ending just above the caput. The appendix is not implicated in any way."

"Coursing with the blood vessels are numbers of shining narrow bands of connective tissue which gradually broaden as they go and end in a slight fan-shaped attachment at various points on the anterior and inner surfaces of the colon. At these points of attachment the gut is held in rigid plication."

In the description of the minute examination of specimen the pathologist specifically states there is nothing whatever to suggest an inflammatory origin.

The observations of Jackson point to the embryonal origin of this structure, particularly because of the regularity of its vascular arrangement and the absence of fibrin, polymorphonuclear leukocytes, or other evidence of inflammation.

Similar bands or veils may form as the result of inflammatory conditions in adult life, but may be differentiated on close observation from these prenatal structures.

Some observers have believed that the mobile kidney plays an important part in the development of adventitious bands in this region during adult life.

Lane,²⁸ in addition to his contention that these bands develop from static pull, also recognized the possibility of their inflammatory origin, since he says: "The inflammation of the cæcum and ascending colon consequent on their excessive distention produces an adhesive process between the outer wall of this bowel and the peritoneum with which it comes in contact. The formation of these adhesions is advantageous, in that they help to support the increasing weight of this portion of the bowel and to some extent to oppose its distension and downward displacement. Besides existing along the outer aspect of the colon these adhesive

processes develop about the hepatic flexure, and by their contraction they draw up the hepatic flexure, exaggerating still further the acuteness of its angle and the condition of obstruction at this point. Precisely similar changes take place at the splenic flexure. After a time these adhesions develop into distinct mesenteries and strong bands."

The relatively greater frequency of ptosis of the hepatic flexure and right colon and its infrequency at the splenic flexure can be explained by its developmental sequence. In the process of development of the large bowel the splenic portion lies at the root of the mesentery and first appears in development, while the hepatic and right colon form later, hence are more liable to irregularities.

There is considerable doubt in my mind of the possibility of contraction in any embryonal veils persisting after birth or even in those of inflammatory origin, except that incident to the immediate contraction of new connective tissue forming in the reparative process which takes place after the subsidence of the inflammation.

Meckel's diverticulum is one of the types of congenital remains which rather frequently takes part in the causation of acute ileus. The usual manner of this action is the persistence of a portion of the omphalo-mesenteric duct (of which the diverticulum is part) as a band connected with the parietal wall. This band with its attachment acts as a snare over which a loop of intestine may fall and become distended to the point of obstruction.

A similar occurrence takes place when a similar long attenuated band occurs from inflammatory processes. This is particularly true as the result of abdominal section and following appendicitis.

Inflammation in Meckel's diverticulum and also in the diverticula connected with the colon and lying beneath the appendices epiploicæ may give rise to bands acting similarly.

The actual differentiation between congenital and acquired bands may not be made with accuracy by the most refined diagnostic methods prior to abdominal section. In many cases even at operation such differentiation is not made.

The suggestive evidence of congenital origin is the persistence of symptoms throughout the life of the individual in the congenital type. The absence of any evidence of acute inflammatory disease either clinically or pathologically on section or necropsy also points to congenital origin.

Jackson's own description of the onset and character of the pain complained of by patients with pericolic veil shows the distress to have a rather fixed date of development. This, in itself, is in favor of the

inflammatory origin of the bands, and, in the opinion of the author, is evidence of progressive inflammatory changes of mild type added upon the congenital anomalous bands.

The clinical symptoms resulting from such structures and the treatment applied for their relief will be considered under Intestinal Adhesions.

PHYSIOLOGY

The peritoneum is in reality the inside covering. In extent it almost equals the cutaneous surface. Viewed in this way it appears to be essentially a protective membrane.

Its surface is quite smooth and is covered with a secretion which permits of the movement of the intestines upon each other without friction. This freedom of motion for the abdominal viscera is essential to the performance of their particular physiological function.

The reparative power of the peritoneum is so great that small wounds of the structure are sealed very promptly. The great omentum is very active in such reparative processes, promptly plugging a hole in the parietes and in the viscera as well. It also envelops a foreign body and covers over local infections. It is claimed by some that this structure forms a protection to the viscera against cold.

In addition to its mechanical function the peritoneum also acts practically as a closed lymph space, the lymph radicles, which are very numerous, having, according to some, a direct communication with its secreting surface. Others deny that the lymphatic channels open directly into the peritoneal sac, but in any event the communication is very close, and the absorptive power very great. Because of this the absorption of exudates or foreign substances within the sac is very rapid, particularly in the upper portions. Under certain conditions it loses this power of absorption, as in tuberculosis, or in connection with large cysts or carcinoma of the abdominal organs, also in ascites due to hepatic obstruction, and that resulting from cardiac disease. On the other hand in certain forms of infection it seems to show an increased power of absorption, taking care of considerable exudates and even of firm adhesions in a remarkably short time. Owing to this property it has in addition to the function mentioned above a marked protective property, encircling, covering, and walling off foreign bodies, pathogenic organisms, and other harmful substances such as gauze, drains, metal instruments, rubber tubes, etc. So marked is its susceptibility that in the days prior to the development of antiseptic and aseptic surgery it was "noli me tangere." But whereas a healthy peritoneum is exceedingly susceptible to injury

and infection, it possesses in a high degree the property shown by all cells, namely the development of the ability to protect itself, and a proper understanding of the causes of reactions to injury and infection and of the measures for its protection makes its invasion by the modern surgeon a relatively safe procedure.

Besides the protective power or immunity of the membrane itself, the lymph glands within its folds provide a further and exceedingly valuable means of protection to the organism. From the blood-vessels, too, are rushed to attack the invading organisms an army of leukocytes which are, after all, one of the most valuable forces in the battle against infection, many times holding the line of defense until the host of bacteria loses its ammunition or poison, and becomes innocuous. Then again the leukocytes perform the function of the medical corps of an army and carry away the dead and dying tissue cells and leukocytes, as well as the dead of the invaders. Finally these same cells, with the proliferating cells of the tissues (peritoneal), take the most active part in the repair of the damage done by the battle and the restoration of the part to the normal. To understand these changes fully one must be familiar with the normal physiology of the blood, the lymph, and the mitosis of tissue cells, as well as their reaction to stimuli of various kinds.

These forces upon the part of the individual for repair must be conserved by the surgeon, both in the prevention of infection and in its treatment. The ability of an individual to protect himself is limited, and so is the number of combat troops which can be thrown into the battle. Good surgery, therefore, demands that these resources must be husbanded, and only such demands be put upon them as may be well within the limits of safety. It is the duty, therefore, of the operating surgeon to prevent in every possible way the contamination of these structures from unnecessary insult, from contact with noxious material, and particularly from active pathogenic bacteria, for these after all are the final determining factors in the development of inflammation of this structure. After the contamination has taken place it is essential to limit its extent and to utilize every agent at one's command to check the propagation of the microbic organism and its chemical toxins. In the discussion upon the treatment of peritonitis the various methods for obtaining such result will be considered.

The basal surface of the peritoneum is exposed to infection perhaps less often than the serous surface, and is much less susceptible to infection, as shown by the manner in which large extraperitoneal purulent accumulations are kept within limits for long periods without invading

the peritoneal cavity. Notwithstanding this fact there are many cases on record where inflammation of the peritoneal cavity has developed from infection in adjacent tissues.

Absorption.—The process by which substances are taken up by the tissues and introduced into the blood and lymph vessels is most important in the processes of nutrition and of metabolism.

The exact methods by which this process is carried on are not yet fully determined, but remain the subject of controversy and future research. Certain well-known physical factors make up an important part of the process of absorption. Osmosis, diffusion, filtration, and dialysis each has a definite rôle.

The property by which gases, brought into contact within a confined space, become mixed is termed diffusion, and results from the movements of their molecules. A similar property of mingling with each other is possessed by liquids, which obey the same laws as gaseous substances. This is a familiar phenomenon in mixing water and milk, or cream and coffee. The process is so simple that it excites no comment. When, however, the two fluids are separated by a simple membrane permeable to the two substances, diffusion still occurs and continues until the liquids on each side of the membrane are of equal density. The term osmosis is applied to this process in the case of water or similar fluids, and dialysis when applied to diffusible substances.

The diffusibility of different substances varies very considerably, and all bodies are classed in two groups, crystalloids and colloids.

The substances belonging to the first group all have crystalline form, and are all diffusible, yet their power of dialysis varies considerably.

In the second or colloid group the substances have no crystalline form, but are amorphous. Usually such bodies have a large molecule and are only slightly or not at all diffusible.

One of the first processes in absorption is based upon this power of substances to intermingle. The ability of the blood and lymph vessels to take up certain substances and to give them off depends upon the property of osmosis, and also upon certain physiochemical processes within the cells which are, as yet, understood only in part.

It is a well-known fact that a heavier (hypertonic) solution will take fluid from a lighter one, while the soluble crystalline substances in the heavier will pass into the lighter (hypotonic) liquid until they are isotonic. These facts are most important, both in physiologic and pathologic processes in the peritoneum, and also in the application of certain methods in the treatment of these pathological processes.

It has been shown that the injection of a hypertonic solution into

the peritoneal sac results in the interchange of fluid and of crystalline substances with the blood until the peritoneal fluid becomes isotonic with the blood.

If, on the other hand, a hypotonic solution is placed in the sac, it also becomes isotonic, some of the salts escaping from the blood-vessels, while the water is taken into the blood stream.

Leathes and Starling¹⁰ in their experiments demonstrated that when hypertonic solutions were employed, more fluid could be recovered from the peritoneal sac after two hours than had been employed. When hypotonic solutions were used less fluid was recovered. They also show that the rate of absorption was higher during the first half hour than subsequently, 39 per cent, while after two hours only 49 per cent had been absorbed. Diminished absorption later is undoubtedly due to an osmotic equilibrium.

It would seem that when the fluid became isotonic with the blood the process of absorption would cease entirely, but actually this does not follow since the process of absorption continues until all of the fluid is removed from the cavity. It does not seem clear just how this is accomplished, but it is a well-known fact that such fluid injections into the peritoneal space causes an increase in the renal output. When the blood takes up an unusual amount of fluid the intravascular tension must be temporarily increased, and the blood flowing through the kidneys causes an increase of the actual, and to a greater degree the relative, amount of water from the blood. There follows a hypernormal concentration of the blood, and a change in the density which makes the blood hypertonic to the peritoneal fluid, so absorption continues in gradually diminishing rapidity until all the fluid is absorbed.

The rate of absorption from the peritoneal cavity depends for the most part upon the character and density of the fluid to be absorbed. Saline solutions are taken up most rapidly. Albuminous fluids are absorbed but slowly, yet blood will in time be taken up. It seems likely that purulent accumulations may be absorbed in time. The process is necessarily slow and uncertain.

Temperature seems to have little influence upon the rate of absorption. Heat to the point of damage to the tissue cells will diminish the power of absorption.

Some claim that denudation of the endothelial surface decreases absorption.⁹ If this is true, it seems to prove the contention of some that there is a vital principle within the individual cell which to some extent regulates absorption.

Fleisher and Loeb⁶ state that absorption is increased in nephrec-

tomized animals, and in those in which the renal vessels have been ligated. Increase of blood-pressure increases the rate of absorption, and because of this action the addition of adrenalin to the fluid increases its absorption.

It seems possible that the pumping action of the diaphragm as claimed by Dybkowski ⁴ is a factor in absorption.

A third factor in absorption is filtration or the passage of a fluid under pressure through a membrane.

In addition to the passage of water and crystalline bodies into the blood stream because of difference in density, the blood, being composed largely of albuminous substance which possesses a feeble power of dialysis, holds its consistence more or less uniform, and to a considerable extent determines the direction of the osmotic current.

The peritoneum being a very thin membrane has a marked dialyzing property, and certain fluids pass readily through it into the blood stream and also into the peritoneal cavity from the alimentary canal. This latter property is shown by the fact that the introduction of fluid, either tap water or normal saline solution, into the rectum while a drain is *in situ* leading to the skin from the peritoneal surface, causes a marked increase in the fluid escaping from the drain.

This property of osmosis is utilized in the treatment of inflammatory conditions of the peritoneum, as recommended by J. B. Murphy. The blood-vessels have not the power of unlimited absorption of material and the rectal instillation is given with the intention of utilizing this fluid to fill up the blood-vessels, to lessen the absorption of noxious material from the inflamed peritoneal surfaces or from the intestinal lumen, and to dilute such toxic material as enters the blood stream.

Apparently the method accomplishes all these objects, at least the general surgical opinion seems agreed as to the value of the proceeding.

Notwithstanding the readiness with which fluids or even sometimes solid particles pass from the peritoneal cavity into the blood stream, certain conditions arise wherein this property seems to be lost. In cases of cirrhosis of the liver, serous tuberculous peritonitis, cystomata of the ovary, and peritoneal carcinoma, large accumulations of fluid within the peritoneal sac remain unabsorbed. The explanation lies, to a very large degree, in the fact that these fluids contain a large amount of albuminous material, which has a low osmotic power; hence they remain unabsorbed.

The older plans of treatment consisted in concentrating the blood by taking a certain amount of its fluid away by saline purgatives so that the water necessary to replace this fluid would be taken up from the abdomen.

The improvement in tuberculous ascites from simply opening the abdomen and draining away the thick non-absorbable albuminous fluid, and replacing it by a thinner and more osmotic transudate from the blood-vessel, is explained by the more rapid interchange of fluids between the blood and the peritoneal cavity.

There is but little doubt that absorption of material from the peritoneal cavity is increased as pressure is increased. The toxic products in acute suppurative processes, therefore, enter the blood with some rapidity. This absorption is undoubtedly lessened by relieving the pressure by abdominal section and the insertion of a drain.

It is generally believed that the absorption from the peritoneal surface is greatest in the upper portion of the abdomen, and based upon this contention George Ryerson Fowler advised the elevation of the patient into the semi-sitting posture to keep the toxic material in the lower part or pelvic portion of the peritoneal sac. The method has proved so satisfactory that it has been almost universally adopted in the treatment of acute inflammations of the peritoneum.

There seems to be an imperfect consensus as to the amount of influence intra-intestinal pressure has upon absorption from the intestine. It seems proved, however, that in the marked distention due to obstruction there is no absorption. It is also diminished in cases of stasis in any portion of the bowel (Enderlin and Hotz⁵). Water is quickly absorbed in the uppermost portions of the small bowel.

There seems to be a selective activity upon the part of the cells. This is shown even in the absorption of salts.

Protein substances are only absorbed when they are broken down and changed into amino-acids or peptids. Neither of these substances is found in the blood coming from the intestine. It appears, therefore, that there occurs a change in the epithelial cells of the intestine which converts these substances into protein molecules, which are perhaps specific to it. That the molecule is specific for the animal seems likely, since foreign protein is not well tolerated.

Fats are absorbed only when the fat splitting enzymes of the pancreatic juice and bile have acted upon them and brought them into liquid form. Fat globules are seen under the epithelium, which is a proof that a synthesis of fat by union of fatty acids and glycerin has already taken place.

Carbohydrates are absorbed as monosaccharids.

The food is practically all absorbed in the small intestine.

Where the amount of detritus is large, as in food containing a quantity of cellulose, it is carried rapidly into the large intestine.

Cellulose is scarcely touched in the small intestine and is only split by bacteria in the colon. For this reason when treating an intestinal fistula food as free as possible from detritus should be given to lessen the waste.

Relatively to the small gut the absorptive power of the colon is small. Water is absorbed, according to some, up to one half its quantity. Under slow rectal instillation more is probably absorbed.

About 20 per cent of sugar is taken up, glucose being the best form. Some split proteins are absorbed but unchanged protein is not taken up at all. Alcohol improves absorption, hence wine and sugar may be added to rectal fluids. Alkalies are probably taken up well.

When attempting rectal feeding the food products should be pretty thoroughly predigested or the results will be unsatisfactory.

With reference to the quantitative absorption of fluids from the peritoneum, G. Wegner¹³ found the surface of the peritoneum to contain 17,182 square centimeters and the skin 17,502 square centimeters of surface. The absorbing power was enormous, equal to 3 to 8 per cent of the entire bodily weight in one hour.

In the presence of an irritant or very toxic substance a corresponding transudation may take place into the peritoneal sac.

Absorption of Insoluble Bodies.—It seems a well-established fact that insoluble substances may be taken up by the vessels of the peritoneum.

The ability to accomplish this was considered to be due to the presence of stomata. Since the more recent authors contend that stomata are not present, another explanation for the entrance of solid substances into the vessels must be sought. Some explain their transportation by incorporation with wandering cells (leukocytes). In consideration of the route of absorption Dubar and Remy,³ Mafucci,¹¹ and Heusner⁸ show that absorption takes place through the great omentum, the lesser omentum, through the lymphatics in the pelvis as well as over the diaphragm, and that both lymphatics and blood-vessels take part. According to Danielsen,² from examinations of lymph from the thoracic duct and of blood, the colloids are taken up by the lymph stream and crystalloids by the blood. These investigations confirm the views of von Recklinghausen¹² in 1863. Beck¹ observed blood escaping from the thoracic duct after the injection of blood into the peritoneal cavity. Hertzler⁷ states that his own observation confirms the assumption that absorption takes place by the blood stream. He also says that "after observing many hundreds of abdomens in which injections of foreign particles have been made he is disposed to say what Treves said about the appendix, 'Nothing is so constant as inconstancy.' . . . The gut

surface is usually relatively but not absolutely free. This is true whether the great omentum is retained or not.

"The great omentum and about the mesenteric root is apt to be the center of the greatest accumulation."

Pain.—Pain is the sensation imparted to the cerebrum as the result of irritating contact with certain nerve fibers. It is one of the protective forces of the organism. It is the first warning of danger in many instances.

The lowest forms of cells have the power of retraction upon irritation or contact. The higher organisms have more highly differentiated power of recognition through the nervous mechanism.

Three views have been expressed in connection with the origin of pain:¹³

1. That there is provided a special conduction apparatus throughout the body which carries the pain impulse.
2. That the sensation of pain results from an overstimulation of the special nerves concerned with touch or temperature or of other nerves of special sense.
3. That it is a special function of the nerves of common sensation which inform us of the condition of our own bodies, of the internal organs as well as of the surface.

The weight of evidence seems to be against the presence of any special pain sense as carried by particular nerve end-organ and fibers. In this the pain sense differs from that of the special sensations of taste, smell, hearing, and sight, since the latter have a special apparatus for conduction of these impressions.

The ordinary cerebrospinal nerves of sensation appear to be able to reveal to the individual certain quite different perceptions.

While the sense of touch is supplied by the sensory nerves all over the body, in certain localities where this sense is particularly well developed there are provided an abundance of papillæ important in carrying out this function. The arborizations of the sensory nerves in the superficial layers of the skin forming the tactile menisci have a very highly developed power of differentiating sensations.

The peritoneal surfaces, particularly those covering the viscera, are not supplied with such a highly specialized nervous mechanism for sensation. This is probably true because of its protected position, there being but little necessity for such function. It is a well-known fact that there is but little contact impression through the visceral peritoneum. Apparently the parietal peritoneum has a greater amount of pain sense than that covering the viscera. This is probably due to the

sensory nerves supplying the superjacent tissue. It is evident that the anterior peritoneum as shown in hernial regions at operation is sensitive to pain. This is perhaps true of the posterior parietal layer, but the impressions are less active, just as ordinary sensations are less active in the back.

There are, however, certain sensations imparted to the patient through the peritoneum, since compression of this structure is promptly felt even when ordinary contact or a needle puncture is not productive of pain. Similarly, pulling upon the mesentery or traction upon the bowel itself gives the individual a painful sensation. Often there accompanies such manipulation a sensation of faintness, shock or depression. This shock sensation is exaggerated around the spermatic apparatus. That the peritoneal surface is not devoid of sensation is shown by the excitement of peristalsis upon contact or the application of heat. It appears that the peritoneum has a heat sense.

The sensation of pain imparted to the peritoneum by irritating substances, as bacteria entering from an intestinal leak, the presence of an acute inflammation, the constriction of the intestinal lumen, is transmitted by the cerebrospinal sensory nerves of the part.

The sense of distention of the intestine with the accompanying colicky pain may take a different direction through the sympathetic system. For the most part, however, sensation of peritoneal structures is conveyed by the ordinary sensory nerves. The location of such painful sensations by the individual, while usually at the site of the causative influence, may frequently be some distance away. For instance, diaphragmatic pain is felt in the region of the neck, due it is thought, to the distribution of the phrenic nerve. The impulse in such cases is not clearly differentiated, but is ascribed to the most sensory portion of the nerve endings. Likewise, gall-bladder pain is referred to the right around to the back and in some cases to the left shoulder.

Painful impressions about the kidney and ureter are often transmitted to the hip and thigh.

The pain in appendicitis is often transmitted to other portions of the abdomen. Tenderness, however, is directly over the organ itself.

Since one of the most useful sensations possessed by man for his protection from harmful influence is pain, the most insidious and deadly diseases are those whose approach is unheralded by painful sensations. Many times, on the other hand, physicians are compelled to refuse the early relief of pain, because an accurate interpretation of this sensation is of aid in the proper localization of the affection and its eradication may jeopardize the life of an individual.

Head has described certain zones of cutaneous hyperalgesia which correspond closely with the areas of distribution of herpes zoster of different portions of the body. He concludes "that in the ganglion certain stimuli must be transmitted from the visceral fibers to those going to the somatic areas, and produce an irritation of these fibers, so that lighter than ordinary stimuli give rise to pain."¹

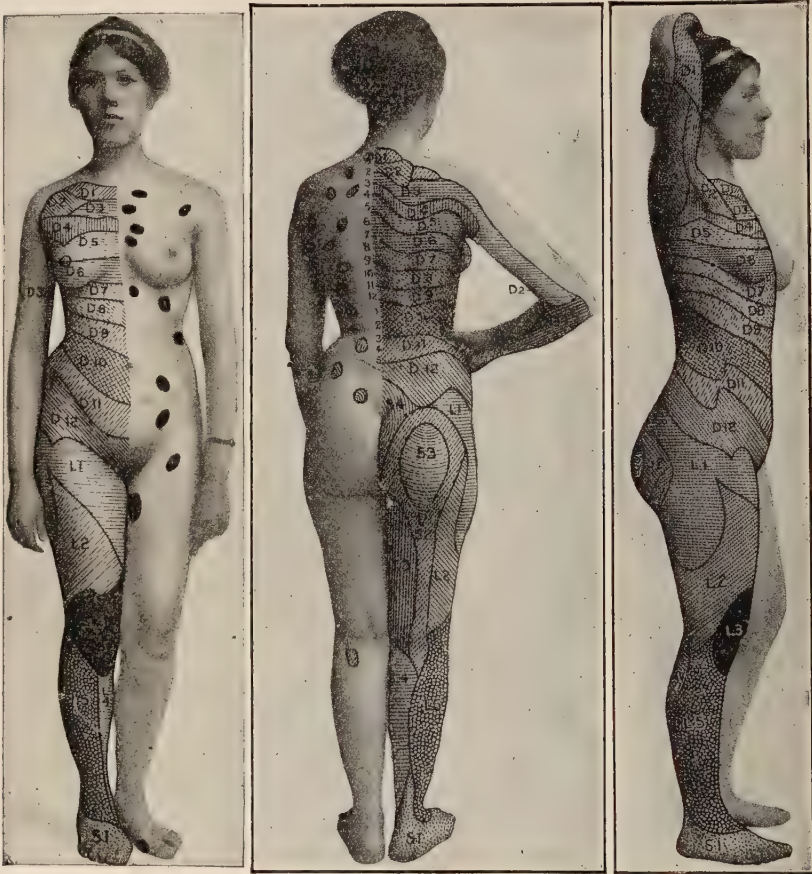


FIG. 23.—ILLUSTRATING CORD ZONES AND AREAS OF MAXIMUM TENDERNESS, ACCORDING TO HEAD.

These zones are shown in the cut which is taken from Behan.

Lennander and Wilms¹⁴ held the view that the nerves in the mesentery, whose function is to supply the visceral peritoneum and the viscera, are less concerned with abdominal pain than the nerves in the parietal peritoneum.

They believe, therefore, that all kinds of abdominal pains are caused by a rubbing of the peritoneal coats or by a pulling of the same membrane.

There seems to be a general unanimity of opinion that the visceral peritoneum reacts but slightly to trauma from the standpoint of pain, but all are agreed that crushing as by a clamp or traction upon the mesentery excites a painful sensation.

It is more difficult to explain the pain in inflammatory conditions, as peritonitis. Part of this pain is due to pressure upon the nerve endings from the engorgement in the peritoneal as well as the other coats of the intestine, gall-bladder or other viscera. A portion is due to distention of the gall-bladder, appendix or intestine. Another portion is produced by peritoneal friction. This is diminished by lessening the respiratory excursion, and there is always a tendency to shallow respiration in peritonitis because of the involuntary effort to protect from pain.

In ileus there is pain because of the mechanical constriction in the internal strangulation, just as in the constriction of a hernia. In addition there is always pain when the arterial circulation is suddenly obstructed from deprivation of nutrition to the tissues. This coincides with Nothnagel's¹⁹ views.

Again, pain is excited when the veins are obstructed and the part becomes distended with venous blood and oxygenation cannot occur.

In strangulation the pain temporarily ceases as soon as necrosis with perforation occurs at the site of constriction and there is relief of intra-intestinal tension as the result of the leak.

In peritonitis, and particularly that excited by ileus, there follows after a short period of freedom from pain a return of this symptom. This pain is due to the direct irritative action upon the nerve endings of bacterial toxins. It is a well-known fact that clean noninfected abdominal or other flesh wounds, when properly approximated, are not painful. But when bacteria enter any wound a burning pain is quickly excited. This is evidenced by cessation of pain when the wound becomes bacteria free.

The pain in ulcerative processes, especially gastric ulcers, according to Kappis,¹² is due to the direct irritative action of the gastric secretion upon the nerves passing to the viscus through the mesentery.

The Sympathetic Nervous System in Relation to Pain.—The older teaching in the physiology of this system was that pain was not excited by stimulation of the normal sympathetic nerves. The more recent investigations of Valentine, Brachet,²⁹ and others show that most severe pain is felt when the sympathetic or its ganglion is the site of inflammatory change, as from exposure to the air for a time.

The fact that abnormal sympathetics are painful forces the conclusion that pain sensations in the abdominal viscera and the visceral peritoneum may be transmitted by the sympathetic nerves.

Rost says^{24a}: "In the final analysis the whole question hinges on the fact that we are ignorant of the kind of stimulus to which the sympathetic responds with pain. May it not be possible that lead, when it produces a typical colic, not only stimulates the motor nerves of the intestines and causes spasm, but also irritates the sensory nerves in such manner as to produce pain." The pain elicited in lead colic and in obstruction as well appears as waves of pain simultaneous with the waves of spasm. It appears likely that pain conduction through the sympathetic is less rapid than through the cerebrospinal system. It probably is more active in producing shock and continues for a longer time than does ordinary painful sensation through the spinal system.

The paths by which the pain stimuli reach the centers are not known. Head has shown, however, that there is close contact in the gray matter of the cord between the sympathetic tracts from the viscera and spinal sensory nerves from the skin. This accounts for the hypersensitiveness of the skin in many diseases of the viscera.

The weight of opinion coincides with what has been stated above. Most peritoneal pain sensations are transmitted through the cerebrospinal nerves, yet certain visceral and peritoneal sensations are carried by the sympathetic.

Rost says^{24a}: "In view of all this, it must be said in summary that even if the pain from mechanical stimuli arises almost exclusively from the parietal peritoneum and mesentery, *i.e.*, indirectly, we are, nevertheless, capable of experiencing certain sensations from pathological processes in our viscera by way of sympathetic nerves arising in the organs themselves."

He quotes Lewandowsky¹⁷ who "also admits that the muscles supplied by the sympathetic system possess sensibility. He (Lewandowsky) assumes that certain stimuli normally reach only the spinal cord, but when increased they may suddenly speed upward and then be transmitted as pain to the periphery. He compares these processes to those in striated muscles from which we ordinarily do not receive sensory impulses, but when an abnormally powerful contraction occurs, as in the calf, severe pain is experienced."

Kramer¹⁵ concludes that pain stimuli, however produced, are carried by the sympathetic fibers, and by communication with the cerebrospinal system, mainly through the vagus and splanchnics and somewhat through the phrenic, are conveyed to the cord and thence to the brain. The pain is evidenced through the peripheral distribution of the spinal nerves.

The views of Kramer appear reasonable and explain the method by which propagated pain is felt in other areas than those in which they are

produced. Certain cutaneous hyperalgesias are also explained in the same way.

The somatic distribution of the segments receiving abdominal and visceral stimuli is responsible for the cutaneous hyperalgesia and the muscular rigidity.

It has been found advantageous to divide the nervous system supplying the organs which function involuntarily into the sympathetic and cranial, that is to say, the sacral autonomic portion. This is a physiologic rather than an anatomical classification. Throughout the vegetative nervous system the fibers do not pass directly from the brain to the organs but enter certain groups of nerve cells, the ganglia. It is claimed that here they are completely freed from cerebral control, but pass as more or less independent nerve structures into the tissues they innervate. In part this is correct, but certain functions which are ordinarily autonomic are to some extent under central control. This is shown particularly in relation to the bladder and the intestine. In certain instances when evacuation of these organs seems imperative it is possible to defer the process by a strong effort of the will. It seems reasonable to conclude that for the most part the autonomic system is free from the control of the will, but that under certain conditions this power which has been delegated to the ganglia of distribution may be resumed by the cerebrospinal system.

A study of the nervous arrangement in the intestine is necessary to a full appreciation of the method of excitation of peristalsis.

The motive power of the intestine is derived from very fine nerve fibers which are supplied to the circular and longitudinal muscular layers. The longitudinal layer has embedded in its substance ganglion cells. This layer is thinner externally and less complete than the internal continuous circular layer. In the colon the longitudinal layer of muscular fibers appears as three distinct bands or tapes of muscular fibers, the *tænia coli*, *tænia mesocoli*, *tænia omentalis*. These bands unite on the *caput coli* at the site of the base of the appendix. This, by the way, is the accurate landmark to locate the appendix at operation. The amount of muscle in the circular layer increases somewhat from above downward according to Roith,²³ Werner, Spalteholz,²⁵ and others. This Rost^{24b} denies, as he claims his planimetric measurements have disproved this idea.

The longitudinal bands in the large gut are in about the same state of tonus (contraction) as the circular fibers. According to Rost^{24c} these tapes act functionally like elastic bands and by tension keep the bowel shortened and also produce the sacculations between the *plicæ semilunares*.

In order to observe the movements of the intestines under physiologic conditions, three methods have been employed. One of these utilizes the

excised organ after a laparotomy, when the structure should be covered with Ringer's or other physiologic solution to preserve approximately the physiological conditions.

The use of x-ray observation is a valuable method because of the normal physiological behavior of the intestine under observation.

The third method is the adjustment of a transparent celluloid window in the abdominal wall.

A fourth method may now be employed by means of a laparoscope.

Cannon⁵ differentiates, exclusive of those of the muscularis mucosæ, the mixing movements and the so-called peristaltic wave. Mixing movements arise from rhythmic contractions of both muscular layers. The simultaneous contraction and relaxation of these layers produces a kneading motion. In addition there are certain so-called pendulum movements from alternate contraction and relaxation of segments of the bowel of varying lengths.^{18, 26}

The author has personally examined these contractions during abdominal sections for intussusception where they were particularly active, and have seen the invagination actually occur. It appeared at these observations that the segments were more or less uniform in length. An interesting study is the determination of the actual reason why the impulse to contract is limited to particular segments while others are relaxed. This apparently points to their nervous supply as arising from certain sections of the nervous system and that the impulse passes to the segments in rotation. It is quite possible that there is an overlapping of supply to prevent dissociation of movements. The exciting cause of these impulses apparently lies within the mucosa, and is probably due to the presence of substances irritating to this structure. The very rapid peristalsis, which occurs when very noxious substances reach the intestinal mucosa, would seem to support this view. The impulse to contract results from the central stimulus obtained from the irritating substance. This action may be reflex, the impulse going only to the ganglion and back, only a slight cerebral notice being taken of the excitation.

The peristaltic wave which carries the contents forward is of this type.

There is an additional movement known as Exner's needle reflex. When the mucosa is irritated by needle puncture contractions of the muscularis mucosæ follow. It bulges out and attempts to push the pointed end downward. Thus, pins which have been swallowed point downward are turned and pass onward head first.^{24c} Open safety pins of considerable size have passed in this way.

This progressive movement begins only when a certain amount of internal tension is reached, according to Trendelenburg.²⁸ The effect

depends also upon the rapidity with which the intestine is filled. "If the filling takes place too slowly, peristalsis frequently does not occur at all, but this approaches the pathological," according to Rost.^{24d} He also states that intestinal paralysis is the result of distention and not its cause. There is, he says, another movement, the so-called "rolling movement or peristaltic rush, in which the contents are forced very quickly through long stretches." In addition may be mentioned the marginal current which may be directed either upward or downward independent of the direction of the central mass.

Considerable discussion has taken place over the question of antiperistalsis and its cause. Some observers deny the possibility of such action. Others, Cannon,⁵ Stierlin,²⁷ Bloch,² Boehm,³ Rieder,²² have conclusively shown that this action does occur.

The intestinal peristaltic movements are dependent upon the nerve supply to the muscular coats. The pendulum movements and those of peristaltic and antiperistaltic activity are dependent on the presence of Auerbach's plexus.⁷

Rost states that "Stimulating impulses come not only through the cranial autonomic or sacral system, but also from the vagus or from cerebro-spinal fibres from the lumbar and sacral segments. Inhibitory fibres which reach the intestines by way of the sympathetic arise in the coeliac ganglion in the superior and inferior mesenteric plexus, that is, directly through the splanchnics.

"Stimulation of the sympathetic leads, therefore, to a slowing of the movements or a relaxation of the intestines."

Stimulation of the vagus³ is not entirely in accord, but it always leads to tonic contractions.

Clinical Significance of Hunger Pains.—William H. Higgins,¹¹ in an analysis of 162 cases of gastro-intestinal and gall-bladder disease, calls attention to the fact that within the span of a few years radical changes have taken place in the conceptions concerning this organ. Ideas that were once accepted concerning it are now discarded, and many established facts of yesterday are crumbling in the light of our present knowledge.

The modern conception of hunger pain is of importance in the study of peritoneal and visceral sensations. For years much of our therapeutics was based upon the acceptance of Haller's theory of the mechanical stimulation of sensory nerves. Pawlow²¹ accepted this view and based his assumption upon the effect of wine as an excitant of hunger.

Subsequent to this period, according to Higgins, it was established by Boldyreff²¹ that the empty stomach was contracted and not atonic as

previously held. He demonstrated that the fasting stomach of man exhibited two types of contractions.

"The first is a tonus rhythm, which disappears when the stomach is full, but is always present when the organ is empty.¹¹

"Superimposed on this tonus contraction there occurs the second type, the periodic powerful rhythmic contractions alternating with periods of relative quiescence. These individual contractions gradually increase in amplitude, and the intervening pauses become shorter, until the climax is reached in a number of very strong and rapid contractions approaching incomplete tetanus. This condition persists until food is taken into the stomach, when relaxation again occurs and the cycle begins again."

It has been proved⁶ that the periods of contraction in the empty stomach are synchronous with the periods of hunger sensations. Each separate contraction is synchronous with a hunger pang.

Carlson⁷ demonstrated that the contractions in an empty or nearly empty stomach give the sensation of hunger by stimulation of sensory nerves located not in the gastric mucosa but in the submucosa or muscularis. The hunger sensations varied with the activity of the contractions. When no contractions occurred the subject did not feel hungry.

Hunger pain is the term employed to describe the sensation felt by sufferers with gastric ulcer. It is excited in the same way as normal hunger sensations except that the sensations are exaggerated. Higgins¹¹ suggests that the contractions in ulcer cases are probably due to hyperexcitability of the gastric nerves which may be explained by local inflammatory processes.

The presence of free acid in the stomach as causative of this pain is upset by the above findings.

Herz¹⁰ and others have shown that the presence of hydrochloric acid is not necessary to the production of hunger pains and probably bears no relation to it.

The history of relief of pain upon taking food followed at varying intervals by recurrence of the pain is strong presumptive evidence of ulcer and a very important point in differentiation of gastric ulcer as a causative factor in perforative peritonitis. Also it is important to recognize the fact that frequently pain of gastric ulcer is relieved by neutralizing the hyperacidity.

Higgins concludes:

1. Hunger pains may arise from normal rhythmic gastric contractions even in the absence of any demonstrable lesions.
2. Hunger pains are not pathognomonic of peptic ulcer but may also occur in connection with chronic cholecystitis and chronic appendicitis.

3. In an analysis of 162 gall-bladder and gastro-intestinal infections, hunger pains were present in 50 per cent of peptic ulcers, 15.4 per cent of chronic cholecystitis and 17.5 per cent of chronic appendicitis cases.

4. The most probable cause of hunger pains is a duodenal reflex resulting either from the absorption of bacterial toxins through the branches of the vagi or from a local inflammatory process in the duodenum. The presence of adhesions in the extragastric lesions is undoubtedly a factor, but is not essential to the production of this symptom.

George Waugh³⁰ states that in 97 of 128 cases in which the transverse colon lay at or below the sacral promontory there was a hunger pain which frequently could be relieved by the assumption of the horizontal position and the ingestion of food. He considered this particular hunger pain to be caused by the downward pull of the loaded colon on its mesentery since it always occurred at four P.M., and in the early morning hours when the ascending colon is filled with food.

"Hunger pain, in duodenal ulcer, does not usually arise on an entirely empty stomach; rather the organ is filled with hyperacid fluid when the pylorus is spastically closed during a hunger pain. If the patient eats, the pylorus opens and the gastric fluid flows out. Probably this hunger pain is only a result of pylorospasm."^{24e}

The observations of Higgins,¹¹ Cannon,⁵ Carlson,⁷ and others appear to show that hunger pains are the result of contractions of the stomach, both in health and disease. The exact cause of these excitations is not entirely clear. The ordinary stimulus to the gastric secretion from taste, sight and smell of food must be an excitant of these contractions and the hunger sense by reflex action. Both under normal and diseased conditions the acid gastric secretion resulting from reflex action may excite the hunger sensation. The gastric contents when alkaline or neutral appear not to excite the pain. Hyperacidity seems clearly to increase the spasm and the pain both in gastric and duodenal ulcers. Symptoms of hunger pain are important in the differentiation of peritonitis coming from perforation of such ulcers.

REACTION TO IRRITANTS

The response of the peritoneum to all forms of irritation is exceedingly prompt. The amount of reaction varies very greatly with the character of the irritant and the length of time it is applied. The most important irritants to be considered are traumatism, exposure to air, thermic agents, heat, cold, electricity, chemical substances, foreign bodies, particularly bacteria. There is a close similarity in these changes. The

reaction from the effects of all these irritants varies more in degree than in kind. In the case of bacterial invasion the process is not localized as that produced by other irritants, but tends to spread widely throughout the peritoneum.

Traumata.—Minor traumatisms will be considered first, because the frequency with which the peritoneum is intentionally opened or is accidentally injured.

Aseptic stab wounds or clean surgical wounds meet simply with the phenomena of repair upon the part of the peritoneum. In the case of a stab wound, as soon as the knife penetrates the peritoneal cavity, there is an entrance of air. The muscular contraction of the abdominal wall which occurs at once acts as an involuntary protective measure, and to some extent lessens the size of the opening. The respiratory movements at the same time tend to force the omentum into the opening and in case it is sufficiently large, through it. This structure presenting in the wound has a tendency to plug it and to exclude the external air. Also the presence of its delicate meshes in the wound tends to some degree to limit the hemorrhage, if not to control it entirely.

The Processes of Repair.—The first change noticeable upon injury of the peritoneum is an agglutination of the serous surfaces to each other. This takes place very promptly after the injury occurs. When a cut or torn peritoneum is coapted by sterile sutures there forms about the line of sutures a layer of coagulable lymph. According to some observers this assumes the form of curved fibers resembling the cables of a bridge in their arrangement.⁷ Under this layer of lymph, mitosis of both the endothelial and connective tissue cells takes place. This process goes on by regular stages to complete repair of the damage. New vessels form in this tissue from a proliferation of the angioblasts. The most perfect example of tissue regeneration occurs in injuries of the peritoneum. The process of repair is rapid because of the great vascularity of the tissue.

In the process of mitosis of the endothelial cells and of the fibroblasts, some of the new cells assume large size and are termed macrophages¹⁴ by some writers. They are called polyblasts by Maximow. Mallory¹² calls attention to a group of cells resembling mononuclear leukocytes on the one hand and endothelium on the other and names them endothelial leukocytes. It is not the purpose of the writer, however, to go into the various theories of cell proliferation in wound healing or into the function of certain cells in the removal of any excess of new tissue remaining after repair is complete.

In the new tissue the cells develop into new connective and endothelial

tissue. A short time after complete repair of traumatized peritoneum has occurred and, in the absence of any foreign material, the tissue cannot be differentiated from normal structure.

Freedom from contamination with bacteria is essential for such ideal repair to take place, since the process varies materially under such circumstances. Healing may be delayed by the presence of microorganisms, and repair does not become complete until the tissues have overcome the bacteria. Processes of healing are also delayed by the presence of foreign material which tends to prolong the irritation.

It is undoubtedly true, as stated by Hertzler⁷ that "ideal wound healing can take place only when there are no foreign substances, in the form of bacteria or dead tissue, to disturb the process.

"It cannot be too strongly emphasized that pathologists have not correlated the modern conception of the relation of wound healing and the coincident reaction to the experiences of the surgeon.

"The cause of this is twofold: in the first place the early processes of healing and bacterial reaction, being both responses to injury, are exceedingly closely related; and pathologists have not recognized how widely divergent the lines become, starting as they do at so small an angle. Surgeons, viewing the process from a greater distance, have long known that wound healing and bacterial inflammation, as observed in the peritoneum particularly, are the antithesis of each other. The whole fabric of abdominal surgical practice is based on the empirical demonstration of the fact that this is true."

Warnshuis and Lampert²⁰ report an interesting case of traumatic peritonitis. A boy, sixteen years of age, sliding down a haystack, fell on a broken handle of a pitch fork. The handle penetrated the right side of the perineum about one inch from the rectum for a distance of six to seven inches. The boy pulled out the handle, lay down for an hour and then walked to the farm house.

A lacerated wound of the ileum was found about sixteen inches above the cecum through which the intestinal contents were escaping. The intestines were covered with exudate and were deeply injected.

In this case two tube drains were placed in the abdominal wound. One was carried through the perineal wound into the abdomen after the intestine had been sutured and the abdomen had been flushed. The patient made a good recovery.

Exposure to Air.—Exposure of the peritoneum to air alone has long been recognized as resulting in damage to that structure. Wegner's experiments with air blown into the peritoneal cavity resulted in decided changes in the endothelium and even the complete substitution of cica-

tricial tissue. He did not, however, observe the development of adhesions as a result.

Walthard¹⁹ found that after exposure of the peritoneum to air filtered free of dust and bacteria, adhesions resulted under perfect aseptic conditions.

It appears that little change takes place in the peritoneal endothelium when exposed to air while the part remains moist. When, however, the exposure is prolonged the surface becomes dry, glazed, and congested. Such surfaces become agglutinated very promptly. This indicates that such exposure during operative procedures should be limited as much as possible.

Reaction to Thermic Agents.—Cold.—The results from the direct application of heat and cold to the peritoneum vary considerably with the temperature as well as the length of the time of contact.

Exposure of the peritoneal surfaces to even moderate degrees of cold for some time results in constriction of the peritoneal vessels and gives rise to the phenomena of "shock." The peritoneum reacts promptly to the local application of cold substances near the freezing temperature. It produces a marked temporary constriction of the vessels, followed by a dilatation. Under ordinary circumstances cold does not reach the peritoneal surface except for experimental purposes.

Hertzler⁸ states: "Ice water injected into the free peritoneal cavity may produce death in a few minutes in amounts not fatal when injected intravenously. Death in this instance must be ascribed to unknown influences, popularly called shock."

Murphy¹⁵ states that in lowered abdominal temperature from the local application of cold, the muscular and nervous elements concerned in absorption are less irritable and peristalsis is less active. This appears to account for the apparent benefit and comfort to the patient which results from the local application of cold.

Cold applied to the surface of the abdomen for a brief period produces a contraction of the visceral vessels, followed by reaction and dilatation when it is promptly removed. More blood, more leukocytes, more nutrient material reaches the part.

Prolonged application of cold to the skin surface produces continuous contraction of the vessels of the internally related part for such a time as the cutaneous sensibility is retained. This action upon the deeper vessels does not appear to be due to the direct action of the cold upon the deeper tissues. It results from the reflex action produced by the cold upon the nerves of the skin.

In order to retain this cutaneous sensibility and incidentally the tonic

effect upon the deeper vessels, some recommend the temporary withdrawal of the cold two or three minutes every twenty or thirty minutes followed by reapplication.

The persistent application of a moderate degree of cold does not seem to produce such a benumbing effect upon the skin as to result in loss of this reflex action, while the persistent use of a great degree of cold may defeat its object by deadening the skin so that the reflex control over the deeper vessels is lost. When this occurs dilatation of the latter vessels takes place and the engorgement is increased. Although theoretically this may occur and while with a degree of cold below freezing the abdominal wall might be frozen through, such result is extremely rare.

From a wide experience in the use of cold applications I can recall no case in which even superficial vesication or sloughing has been observed.

One of my colleagues reports that he made such an observation.

The intermittent employment of heat and cold alternately over an inflamed appendix or peritoneal inflammation does not appear logical.

The beneficial effects of cold or heat lie in the tonic contraction of the deep-seated vessels. When such contraction is obtained and the circulation improved thereby this tonic action may be kept up for some time without relaxation and engorgement of the part. A change to heat following such degree of cold produces a marked reaction and the paralyzing effect of both the heat and the cold is obtained. As the result of such action the vessels dilate, become engorged, finally choked with blood and complete stasis and necrosis may follow.

We admit that there is a limit to the amount of cold a part may stand, particularly where great tension is present, such as occurs in the finger in phlegmonous thecitis, and there is great danger of necrosis. Under such conditions cold should not be employed or should be used with great caution.

Similarly in abdominal conditions there is a limit to the amount of cold which may be applied to the surface. If the skin becomes darkly congested and the abdominal wall densely hard the cold is too intense or has had too prolonged application. Under these conditions it must be promptly withdrawn for a time to permit the part to regain its normal tone.

For many years it has been employed in the author's clinic in the treatment of acute appendicitis and acute peritonitis with marked benefit. During this period although the continuous method has been the one usually employed no case has shown any ill effects from its use. The only time the ice bag has left the patient's side in these cases is for the time necessary to refill it. Often the application has been kept up for

seventy-two or more hours. It is well in applying cold for a long period of time to place the ice bag within a flannel sack or to put a towel between it and the better than side skin.

Heat.—That the peritoneum is sensitive to heat is shown by the peristaltic waves excited by its application, which appear to be more exaggerated than those from simple contact.

This does not correspond with the views of Lennander¹¹ or the later observations of Broese,³ who corroborated those of Lennander. Both these observers hold that the peritoneum is insensitive to heat and cold as well as to pressure and pain. While it is quite possible that there is no cerebral impression resulting from application of heat to the peritoneum, there must undoubtedly be a reflex action which is shown by the vascular changes and the muscular contraction. The effects upon the blood-vessels are familiar to all. This is particularly shown by the prompt response to the application of heat to coils of intestine which are discolored as the result of strangulation. In viable structures there is an almost immediate restoration of color, the part changing from purple through a mottled red to a bright pink. When the vessels are thrombotic such a response is slow and imperfect. The examination of such vessels will show the futility of further use of heat, etc. The separation of the serosa from the subjacent structures or failure to regain the normal color after the application of heat for fifteen minutes indicates that the circulation will not be restored. If any portion of the vascular supply fails to clear up, it is unsafe to replace the loop within the abdomen. The coil of damaged intestine should be resected or left outside to be dealt with subsequently.

In applying heat to the peritoneal surfaces care should be exercised not to damage the tissue by excessive heat. Heat which the hand will stand comfortably will be safe for the peritoneum. The application of moderate degrees of heat to the normal peritoneum produces an increase in the vascularity of the part which assumes a pink or reddish hue. Higher degrees of heat result in exfoliation of the endothelium or in superficial necrosis. Moist heat is better borne by the peritoneum than dry heat.

The actual cautery has been used for the cauterization of the stump of the appendix, also in resection of the stomach and other portions of the alimentary canal with the object of obtaining sterility. The results of the experimental work on the use of the thermocautery have not been uniform. Adhesions were found to result from its use by Spiegelberger and Waldeyer,¹⁶ V. Dembowski,⁵ Franz,⁶ and Maslowski.¹³ Baisch² and Keltenborn⁹ were unable to confirm this work. Ten Brink¹⁷ was able to produce adhesions only when infection was present. Küstner¹⁰ found no adhesions at an operation performed fourteen months after

many pelvic adhesions were severed by the actual cautery in the removal of an ovarian cyst.

Vogel¹⁸ claims that superficial burning of the peritoneum results in the formation of adhesions while deep charring does not so result, the eschar remaining as a protective covering until healing of the tissue is complete. Webster²¹ confirms Vogel's findings.

The weight of opinion seems to warrant the use of complete charring if the cautery is used at all. The writer would not hesitate to use the cautery upon peritoneal tissue when indicated. Clinically, however, it has been found that its use in intra-abdominal operations has been rarely necessary.

Local application of heat to the surface of the body in the treatment of visceral lesions is at times very valuable.

Very hot applications to the skin act similarly to cold, causing contraction of the deep vessels. The effect is of shorter duration and only occurs when the heat is sufficient to cause constriction of the cutaneous vessels. This gives way to dilatation especially of the veins from action upon the sympathetic ganglia of the small vessels.

If the heat is kept up at the same degree the constriction of the deep vessels is continued as in the use of cold. The superficial vessels are dilated and the part appears rosy. The difficulty in the application of local heat is to keep it at the same temperature.

Moderately hot applications generally produce congestion of associated vascular areas. This form of moist heat is used to favor the localization of forming abscess.

Both cold and heat are grateful to patients suffering from acute appendicitis and acute peritonitis, and our experience leads to the conclusion that cold is preferable to heat in these conditions.

Very concentrated applications of high degrees of heat to the abdomen accidentally result in a burn. In order to produce a burn of the peritoneum through the abdominal wall the degree of heat is so great that extensive sloughing of the abdominal parietes occurs. It is safe to say, therefore, that such burns involving the entire thickness of the wall including the peritoneum are exceedingly grave injuries.

Injuries from Electricity.—These injuries rarely involve the abdomen alone. When the electric discharge is sufficient to damage the abdomen materially it may prove immediately fatal. Occasionally a burn of the abdominal wall occurs.

Such a burn produces discoloration of the tissues, varying from an irregular red line on the skin to extensive charring. The odor of charred flesh is present. Such a burn has but little tendency to heal. The nervous

mechanism of the vessels is impaired and the tissue cells lose to a considerable extent their power of reproduction.

It takes considerable time for the separation of the sloughs in such injuries because of the action of the electricity upon the tissues. Months may elapse before healing is complete. Rarely are the peritoneal structures involved in such an injury.

The treatment consists of the local application of palliative measures, with restoratives and supportive measures for any constitutional symptoms.

In cases which heal slowly it is well to excise the entire burned area provided only skin and fascia are involved. This may be done with immediate suture, provided the incisions are made in healthy tissue and there is not too much tension to permit coaptation of the wound edges.

Chemical Agents.—Chemical irritants may be considered as gases, solids and liquids. Gases are rarely brought into contact with the peritoneum. Bainbridge ¹ employed oxygen gas in the cavity for the purpose of preventing adhesions. More recently both oxygen and carbonic oxid have been injected into the cavity for the purposes of roentgenological diagnostic studies, and no damage of the peritoneum appears to have resulted.

Walthard ¹⁹ tested the effect of air, oxygen, carbonic oxid, and nitrogen upon the peritoneum and found that none of them produced adhesions as long as moisture was present. Both adhesions and macroscopic changes in the peritoneum resulted from dry air at 38° C. A necrosis of the endothelium resulted from the prolonged contact of dry warm air. Contraction of the blood-vessels, diminished nutrition, and adhesion formation followed this application. The vapor of ether appears to produce some irritation and a tendency to adhesion formation. The more noxious gases produce more extensive destruction of the peritoneal tissue. Formaldehyd gas produces hardening, dryness, and destruction of the endothelium. The same is true of chlorin.

The effect of chemical substances upon the peritoneum was extensively studied in the period of transition from the antiseptic to aseptic methods of handling wounds. Delbet, Grandmaison, and Bresset ⁴ showed by their experiments that the larger number of chemical solutions such as carbolic and salicylic acids, bichlorid and biniodid of mercury were harmful. The effect of iodoform and salol, in their experiments, were found to be less irritating. Hertzler ⁷ claims that iodoform was a frequent cause of adhesions when used during this period. It has been completely discarded.

Caustic alkalies and strong acids act upon the peritoneal structures

just as they do upon all tissue, causing when applied in concentrated form destruction of the cells. This change takes place through the whole area of application. Fortunately, only rarely is such contact made as the result of accident. When the surface of the body is exposed to chemicals of this class the tissue destruction is so great in most instances as to destroy life before the peritoneum is reached. Carbolic acid if left for a considerable length of time in contact with the peritoneum destroys the cells. When it is applied momentarily and is followed by alcohol and then sponged off, it destroys bacterial growth. Many surgeons use it to sterilize the stump of the appendix.

Alcohol is perhaps the least harmful of chemical agents. In 60 per cent dilution it causes minimum amount of cellular destruction, but some abrasion results and necrosis may follow the use of 96 per cent strength. Ether has been employed within more recent years with a view of overcoming bacterial growth in cases of peritonitis. Ether has an anesthetic action upon the cells, but like alcohol tends to harden them and the proof of its irritating effect seems to be established.

Tincture of iodine applied in the treatment of appendicitis and peritonitis, as recommended by Johnson and Crisler, of Memphis, as well as others, has attracted considerable attention. The writer has not been able to concur in their point of view. Even in dilution of half the normal tincture (to $3\frac{1}{2}$ per cent) it is an irritant and causes superficial cell necrosis and a marked tendency to the formation of adhesions.

The least noxious fluids which may be employed in the abdomen are normal solutions of chlorid of sodium and citrate of sodium.

Formalin and chlorin solutions produce hardening of the cells, and result in the formation of adhesions.

We are firmly convinced that the peritoneum is in much better condition for resistance of infection and for repair when it is not brought into contact with chemical substances.

REACTION TO HEMORRHAGE

The peritoneal tissue reacts to hemorrhage somewhat as it does to any foreign substance. As a matter of fact blood is not normal to the peritoneal sac and is, therefore, a foreign substance. The local reaction to the presence of foreign blood of the same species is more marked than to the blood of the individual himself. It is a well-known fact that when blood escapes from a vessel either into the air or into connective tissue spaces, it shows a decided tendency to coagulate.

In addition to the ferment liberated from the injury to the endothelial

cells of the vessel there is also present a similar ferment in muscle tissue which tends to result in coagulation. Notwithstanding this fact some hematomata in the soft tissues consist of part coagulum and part fluid blood. This may result from the large amount of blood accumulating in a short time, and only sufficient fibrin ferments being present to coagulate a portion of the whole.

In the serous cavities while there is also present the same tendency to coagulation it is much less marked than is the case where the blood is exposed to the air or when it occurs among the connective tissues. In the latter tissues the amount of extravasation is somewhat limited by the tension present in such part. In the large serous cavities the amount poured out in a given time is usually larger and the resistance it must overcome is less. These conditions may to some extent delay its coagulation.

In addition there appears to be something in the secretion of the endothelial cells of the serous surfaces which tends to keep the blood fluid for longer periods of time than is the case in other localities. The exact nature of this antithrombotic substance is not known at this time. The delay in coagulation does not occur in hemorrhages occurring within the skull where the greater portion is always coagulated. A small amount only is fluid under these conditions when there is an active hemorrhage present. If the hemorrhage into the cranium has ceased the blood promptly coagulates. Therefore, it would appear that there must be some difference between the action of the cells of the meninges and the cells of the pleura and peritoneum to hemorrhage.

In view of the fact that in cases of hemorrhage into the serous cavities it has been proposed to employ the extravasated blood for auto-transfusion, it appears that the question of changes taking place in the character of such blood should be carefully determined. If hemolysis is likely to take place following the injection of such blood into the vessels of the same individual it becomes evident that this procedure could not be harmless. As evidence that hemolysis does occur as the result of the reabsorption of blood from the peritoneal cavity, it may be cited that in all these patients there is noticed a peculiar icteric tinge to the skin which is believed to be due to the absorption of the products of hemolysis and is often so marked as to be diagnostic of internal hemorrhage. The French writers upon this subject claim that the blood coagulates *in toto*. Riedel³ and others have found that rapid absorption occurs without coagulation.

It seems quite possible that blood can be rather readily taken up from the peritoneum before coagulation occurs. Undoubtedly the serum ex-

pressed during coagulation can be absorbed even more readily. It is also well established that in time considerable clot may be taken up by the absorbent vessels. Penzold² found that when small amounts of blood were introduced into the peritoneal cavity no blood was found at the end of three days. He concludes that because of this finding the blood must have remained fluid for some time. The greater part of such blood probably was absorbed very soon after entering the cavity, but even clotted blood might be absorbed by an active peritoneum in three days when the amount was small and no serious injury interfered with its power of absorption.

None of the observers appears to have considered the possibility of serum escaping into the peritoneal cavity and being in part responsible for its fluidity. There is no evidence to show that such a process may not take place. It is quite likely that the vessels will take up in a given time as much of the damaged blood as may be safely cared for, and if so the methods of autotransfusion recently suggested are to be adopted with a considerable degree of caution.

Penzold says that changes in the sense of a peritonitis were not demonstrable. This is probably true in the early hours after an intra-abdominal hemorrhage; later there is considerable reaction, sometimes inflammation and even suppuration is present. These results, however, follow contamination of the blood.

Schrunder⁷ has shown that blood of itself does not tend to cause the formation of adhesions. It is certain, however, that the addition of bacteria to the blood in the abdomen certainly does tend to the formation of adhesions, which is the final result of the process of repair as well as that of a subsiding inflammatory process.

Rost⁶ states that hemorrhage into the peritoneal cavity is a powerful irritant and leads to peritoneal shock. "The clinical picture in such intra-peritoneal hemorrhage is apparently very serious. Actually, however, the danger from hemorrhage alone is not very great. Even when patients appear exsanguinated after injuries to the liver or spleen, there may be found only a small amount of blood. Many gynecologists have recently become more conservative in the treatment of extra-uterine gestation."

In part this contention is true. There is always clinical evidence of intra-abdominal hemorrhage even in moderate amount. Concealed hemorrhage is always serious since the amount of blood loss is impossible of correct estimation until brought into view by the surgeon. The systemic reaction to hemorrhage is always considerable, particularly that occurring into the abdomen. Some serious visceral injuries without

hemorrhage may show but little shock, but when both are present the shock is considerable.

It has been positively shown that hemorrhage into the peritoneal sac is productive of irritation, since the clinical picture is quite similar to that of peritonitis. Abdominal tenderness, rigidity, and pain are evidences of this fact.

Effects of Extravasated Blood.—This accident may occur as the result of traumatism, from rupture of an ectopic gestation sac, from rupture of hematoma of the ovary, from rupture of the uterus, and following incomplete hemostasis during operation. Massive hemorrhages give rise to such alarming symptoms that immediate treatment is demanded, and the symptoms, prognosis, and treatment are the same as for hemorrhage.

More particular interest surrounds, in this connection, those more gradual accumulations of blood and the local reaction of the peritoneal structures to its presence. Blood in the peritoneal cavity usually coagulates, but in some instances a considerable portion may remain fluid.

Minor hemorrhages may be disposed of in a short time by the absorbent vessels. The larger and even some of the smaller extravasations of blood may remain and act as a foreign body. There is always a local reaction of the peritoneal structures with the formation of coagulable lymph on the surface which cements the coils of intestine to each other and agglutinates the mesentery so as to isolate the clot. The amount of lymph outpoured is proportionate to the amount of clot. The reparative process is so stimulated that part of the blood is taken up. Usually, however, this process is incomplete and some of the bacteria present within the intestinal tract, or more rarely a colony floating in the blood stream, invades the clot. An inflammatory process is then excited, which usually results in suppuration. The local changes are identical with any inflammatory process—the abscess either empties itself into one of the hollow viscera, or occasionally may point on the surface, particularly at the site of an operative wound, or it may be relieved by incision and drainage.

Symptoms of Intra-peritoneal Hemorrhage.—Pallor, thirst, syncope, restlessness, pain, rigidity, rapid and feeble pulse with diminished pressure in the vessels are present in this condition. If the bleeding is slow or intermittent the pulse will rally, and again become weak from time to time as reaction seems about to set in. This is a very characteristic symptom. The examination of the blood will show a reduction in the number of both the red cells and of the leukocytes. The relative proportion remains the same.

The percentage of hemoglobin is diminished proportionately to the amount of blood lost because of replacement of the blood by water from the tissues. If a large quantity of saline solution is placed in the veins there may be a still further temporary reduction. The color index may remain unchanged or show little variation. It may be lessened. The volume of the blood is restored by the large quantity of water taken into the vessels. After the hemorrhage is controlled the leukocytes show a relative increase because their reproduction is less complicated and more rapid than that of the red cells.*

French¹ states, in discussing anemia: "It is sometimes stated that the result of blood-loss is to produce an anæmia in which the red corpuscles and the hæmoglobin are equally reduced, so that the colour index remains more or less normal. This may be true of an acute bleeding such as venesection or post-partum hemorrhage, but the effect of recurrent blood-loss is to produce the chlorotic type of anæmia, in which the red corpuscles are less diminished than is the hæmoglobin."

Stitt⁸ in speaking of color index in anemia states: "In anæmias we have three types of color index: (1) The pernicious anæmia type, which is above 1. Here we have a greater reduction in red cells than we have of the hæmoglobin content of each cell. For example, in a case of pernicious anæmia we have 2,000,000 red cells (40 per cent) and 60 per cent of hæmoglobin, $60 \div 40 = 1.5$. (2) The normal type, when both red cells and hæmoglobin are proportionally decreased, as in anæmia following hæmorrhage. (3) The chlorotic type. Here there is a great decrease in hæmoglobin percentage, but only a moderate decrease in the number of red cells. Hence the color index is only a fraction of 1. For example, in a case of chlorosis we have 40 per cent of hæmoglobin and 4,000,000 red cells, $40 \div 80 = 0.5$.

"One can judge fairly well the approximate color index by noting the character of the staining of the red cells. This is faint in bloods of low color index and deeper than normal in cells in a case with high color index."

The usual belief that a high leukocytosis is found after hemorrhage is probably the result of observations taken during an active process of repair, in which there is always an increase in the production of leukocytes.

Rose and Carless⁴ state: "If the blood is examined immediately after a patient has suffered from a severe hæmorrhage, it will naturally be found to be normal in composition; part has been lost, but the quality of the remainder has not altered. After a short time the volume of

*The observations above recorded are the result of the author's own experience.

blood is restored to normal by means of fluid derived from the tissues. At this stage the blood is more diluted than normal, the red corpuscles and hæmoglobin being alike reduced, so that the colour-index remains 1. There is also in most cases a temporary increase in the number of leucocytes."

They ⁵ also state: "During the continuance of hæmorrhage the blood-pressure necessarily falls; but unless a volume equal to about a third of the total bulk of blood in the body is lost, it quickly rises again to the normal after the bleeding has ceased. This rise in blood-pressure is partly due to a diminution in the size of the vascular area, owing to vasomotor contraction of the peripheral arterioles and of the splanchnic area, but is also caused by an increased flow of lymph into the circulation."

Prognosis.—This depends upon the amount of blood lost, the suddenness with which it escapes from the vessels, and upon the resistance of the patient. It is always a serious, and often a very grave condition. Proper arrest, with measures to counteract shock, and transfusion make the chances of recovery much better.

Treatment of Peritoneal Hemorrhage.—This must be conducted upon the same general lines as the treatment of hemorrhage in any locality. If the hemorrhage has ceased when the case comes under observation and the evidence of this fact is conclusive, noninterference is permissible. The patient is placed at rest, and any existing depression is overcome by the judicious and careful use of stimulants. It must be borne in mind that if the activity of the heart is much increased a recurrence of the hemorrhage may be excited. Morphin acts both as a sedative and as a heart tonic and the amount of stimulation is not so marked that hemorrhage will be excited. It has quieting but bracing action on the heart. Ice may be applied to the abdomen to control hemorrhage. It should not under these conditions be alternated with heat.

Because of the uncertainty as to whether concealed hemorrhage will continue or not, the weight of opinion is that the abdomen should be opened whenever the assumption of the presence of hemorrhage is strong. When a vessel is bleeding it should be controlled. When this is accomplished the volume of fluid in the vessels may be restored by injections of normal saline or glucose solutions or, as is better in the more severe cases transfusion of blood. The donors should be properly typed and be healthy individuals. In the less grave cases one pint of normal saline with one dram of adrenalin solution, 1 to 1000, may be given by Murphy drip with good results.

The patient must be kept warm. If severely shocked and the surface is cold a dose of 1/150 grain of atropin sulphate will dry the skin

and bring warmth to the surface without heat loss. Heat to the surface is indicated. The patient should then be placed at rest. To this end morphin may be used safely.

HEMORRHAGE FROM ECTOPIC GESTATION.—One of the best examples of intraperitoneal hemorrhage is that occurring from rupture of an ectopic gestation sac. A tubal abortion also produces considerable hemorrhage, lasting perhaps over long periods of time. The amount of blood lost in these accidents is large.

Rapid anemia, marked pallor, profound shock, air hunger, and cold extremities are all evidences of this form of internal hemorrhage. The skin becomes icteric in a short time if the case does not promptly terminate fatally. The mortality records obtained from the coroner's office in Philadelphia a number of years ago demonstrated the frequency with which death followed this accident at that time.

Lawson Tait first operated for the relief of this condition in 1883. Price showed a remarkable series of recoveries following prompt operation for the control of the hemorrhage and the relief of the patient.

The reaction of the peritoneum to the presence of blood in these cases is most interesting. The incidence following rupture of a tubal gestation sac shows either the immediate loss of a large quantity of blood with shock, prostration and death; the loss of a moderate amount of blood with a considerable hematocele, or a small blood loss with small hematocele. The latter may be quickly absorbed and in time the dead fetus will also be absorbed if early in the impregnation. The large hematocele, however, may be taken care of after a long period of convalescence. It may, however, become infected and the woman have a very stormy voyage to a port of safety, if she reaches it at all.

The local changes within the peritoneum consist in the coagulation of a large amount of blood. The serum which is freed by this process is promptly absorbed, taking the place of part of the fluid lost from the blood-vessels. The peritoneum reacts to the presence of the blood by the agglutination of the coils of intestine adjacent, while the omentum caps the mass on top. The capillaries in these structures are increased in size and there is perhaps an outpouring of fluid into the peritoneal cavity. At least, the normal peritoneal fluid is increased in amount. This tends to hold some of the blood in a fluid state. The leukocytes are increased in the vessels and at the point of hemorrhage they become phagocytes, incorporating portions of the clot into their structure. In the absence of bacterial contamination the process finally progresses to complete absorption of the clots. Certain portions of the clot may remain longer and favor the formation of adhesions, which in time disappear.

This process of absorption takes place rapidly at first, but the rate of absorption is diminished so that the last portions require some time in their removal. The writer has often observed large clots remaining for more than ten days after the accident has occurred, all hemorrhage having been checked for some time before the operation.

A very interesting study is the manner by which the peritoneum adjusts itself to the presence of the fetus. In cases of sudden and total rupture of the tube and escape of the embryo into the peritoneal cavity, the likely termination is death of the fetus. The blood clot surrounds the embryo and under these circumstances the fetus is absorbed together with the blood clot if the accident has occurred during the early weeks of gestation.

In certain cases of tubal rupture and more particularly of tubal abortion, the embryo may remain alive, but only in cases where there remains a more or less perfect placental attachment, albeit this connection may be tubal, ligamentous, peritoneal or even more distant (J. W. Williams). Its development may continue until the end of normal period of gestation, and a living child may possibly be delivered by an abdominal section. Death of the fetus may take place at any period during this time and the embryo become encysted and remain as a lithopedion or assume the characteristics of a dermoid.

In some cases fetal bones are extruded one at a time, usually through the rectum, or perhaps the vagina. The power of the peritoneum to take care of the situation is remarkably good.

The recognition of the presence of an ectopic pregnancy is based upon a gradual enlargement of one oviduct without a corresponding increase in the size of the uterus. The os is soft and velvety as in normal gestation. There is the typical history of a missed period followed perhaps at each month by cramps and a small amount of bloody flow. Some shreds may pass. The breasts are enlarged. Nausea and vomiting may be present. When, following such a history, the woman is seized with sudden intra-abdominal pain, pallor, and shock, the diagnosis may be safely made of ruptured ectopic gestation.

Treatment of Hemorrhage from Ectopic Gestation.—The treatment of ectopic gestation has received considerable discussion during recent years.

Some observers have adopted the view that where the patient has passed the immediate hemorrhage safely, she may be permitted to wait some days before a section is done. This stand does not seem tenable and the reasons seem very cogent. If the patient is seen immediately after the rupture has occurred, a bleeding vessel is to be controlled. There is no

positive way in which the amount of hemorrhage can be determined, nor can the certainty of its complete stoppage be assured.

It appears imperative, therefore, to control the bleeding permanently. This can be accomplished only by abdominal section and visual ligation of the bleeding vessel.

The mortality is so very slight following such procedure in competent hands that the danger from interference becomes negligible. Careful cleansing of the abdominal cavity is the principle to be followed, but this should be done by aspirating the extravasated blood by intra-abdominal suction rather than sponging. Transfusion has not been necessary in a large series of ruptured ectopics, but, nevertheless, if the condition of the patient is very poor, it should be given regardless of whether or not the extravasated blood is allowed to remain.

The operative management of a viable ectopic pregnancy at term by abdominal section may bring a living baby. The management of the placenta in such case may be accomplished by closing the abdomen without a drain and depending upon the placenta being removed by absorption.

In cases in which the fetus is dead, it may be better practice to marsupialize the sac and employ drainage to take care of the embryonic tissues. There is considerable danger in the forcible removal of a closely attached placenta.

AUTOTRANSFUSION.—This term is applied to the replacement of blood which has been extravasated into the peritoneal or pleural sac into the vessels of the patient. The procedure was first employed by J. Thies³⁵ in 1914. It has been used most frequently in Germany. Lucius E. Burch reported upon its use before the Southern Surgical Association in 1922,⁶ having collected 164 cases in the literature, of which only 4 cases were reported outside of Germany. He arrives at the following conclusions:

“Autotransfusion is a safe procedure, although in a limited number of cases reactions will occur.

“Sodium citrate is not essential. Normal salt solution will make an admirable substitute and if neither of these is at hand, the pure blood may be re-injected.

“Extra-uterine pregnancy will offer the largest field of usefulness for this procedure, but in wounds of the spleen, liver, wounds of the lung producing a hæmothorax, and in operations where a large amount of blood is unavoidably lost, it will not only save life but will hasten post-operative recovery.

“Contaminated blood should not be thrown away, but should be given as a rectal drip.

“Autotransfusion may occasionally be used to advantage in certain

obstetrical complications, such as placenta prævia, rupture of the uterus, and Cæsarean section."

Burch gives the history of a man fifty-one years old upon whom a splenectomy was done. The large amount of blood lost within the abdominal cavity was recovered by expressing the contents of the abdominal packs into a sterile glass receptacle. Some 800 c.c. of blood was obtained in this way. After it was citrated and strained it was injected into a vein at the elbow. There was no reaction, the operative recovery being smooth. The method employed in this case was simple and the technic easy of application. The necessity of using large packs facilitated the recovery of the extravasated blood.

When an automatic electric or hydraulic pump is at hand the blood may be taken up in an aseptic way and at once conveyed into a receptacle containing either 2 per cent solution of citrate of sodium or normal saline solution. This facilitates the handling of the blood and the completion of the transfusion with the least exposure to the air and the smallest chance for contamination.

Eberle⁹ and also Wederhake³⁷ recommend the use of the aspirating needle in autotransfusion in thoracic surgery. The same instrument may be employed for abdominal cases. Murray B. Davis⁷ reports a case of autotransfusion for gunshot wound of the spleen, which met the indications well. The fact that the patient died on the ninth day from bronchial pneumonia does not mean that this was the result of the transfusion. Blechschmidt² reports four cases of autotransfusion done for ruptured tubal pregnancy. Some of the transfusions are followed by considerable reaction. Bumm³ reports two cases of thirteen in which rigor and fever followed the transfusion immediately. He considers the patient's own blood is superior to any other donor. Doederlein⁸ takes the position that autotransfusion is a safe and harmless proceeding.

Apparently the greatest danger in autotransfusion is the contamination of the blood taken from the abdomen with bacteria. Theoretically in the use of blood taken in cases of ruptured tubal gestation there is always a possibility of contamination, but Thies³⁵ states that the blood was free from bacteria in his three cases. Toepler³⁶ reports reinfusion in 24 cases of ruptured ectopic gestation, and quotes Sweitzer as reporting one death from hemoglobinuria. From this report it would appear that hemolysis is possible and that the method is not altogether devoid of danger. It would seem from a study of the literature that recent profuse bleeding from rupture of the spleen, liver, or a large vessel may be benefited by autotransfusion. It is dangerous to take blood for this purpose from the

abdomen in which an intestinal wound permits fecal escape because of possible bacterial contamination and coagulation as a result.

The writer concludes from his own experience with ectopic gestation and the reports of Mordecai Price and others, by whom as many as one hundred sections for ruptured ectopic pregnancy have been performed without a fatality, that the use of autotransfusion in such condition will be likely to produce a higher mortality than will the operation alone as usually performed. We disagree with Burch⁶ who states that contaminated blood should not be thrown away, but should be placed in the rectum by the drip method. It will do no more thus given than will saline or glucose solutions.

INTRAPERITONEAL TRANSFUSION WITH CITRATED BLOOD.—In a previous paper Siperstein³⁴ found the use of citrated blood for intraperitoneal transfusion was a safe procedure, simple in application and a method of distinct value. He reports 5 cases and reviews the literature at length. He could find no report of the clinical use of the intraperitoneal route for transfusions since 1884. So far as he knows, citrated blood has never previously been given intra-abdominally. He employed the following method: Patients were allowed no breakfast on the day of the transfusion; no other preliminary precautions were taken. A freshly prepared 2 per cent solution of sodium citrate is used in the proportion of 10 c.c. of solution to every 100 c.c. of donor's blood. Both donor and recipient are grouped before each transfusion. Under strict asepsis, the donor's blood is strained through gauze and immediately injected into the peritoneal cavity of the recipient.

Among the 5 cases recorded, 3 showed favorable results. He claims the method to be simple and useful when other routes for transfusion are unavailable or impractical.

REACTION TO FOREIGN BODIES

The effect of foreign substances upon the peritoneum has been repeatedly observed. Foreign bodies placed within the cavity under aseptic conditions may remain for some time, in many instances, without exciting more than a local reaction. Aseptic dead tissue such as that produced by the actual cautery or that destroyed by chemical or mechanical means may be completely covered and surrounded by the peritoneal exudate, and while the reparative process proceeds to completion in the adjacent tissue in which the cells are not devitalized, the dead cells are gradually removed and finally the reparative process goes on apace. Large denuded areas of peritoneum are very promptly covered by new granulation tissue and subsequently by true peritoneum in the reparative process.

When a foreign body, as sterile cork, a piece of sponge, a linen or silk thread is left within the peritoneal cavity, either intentionally or by accident, the reparative forces of that tissue are immediately called upon to take care of it and to render it innocuous. Almost at once there is a local reaction with an increase of the albuminous material put out by the cells of the membrane at the point of contact. There also occurs an engorgement of the blood-vessels, which further increases the exudate. The peritoneal surfaces grasp and more or less completely surround the object. In cases of gauze or sea sponges, there is an ingrowth of the peritoneal tissues into the interspaces of these bodies. A considerable amount of fluid is poured into the sponge or gauze, with the result that there is a gradual pressure of the foreign substance toward the point of least resistance, depending upon the location, either toward the abdominal wound, if superficially placed, or toward the lumen of the large bowel or vagina, if lying in the pelvis. The amount of local reaction depends upon the size and character of the foreign body and upon the presence or absence of infection. Even in the absence of infection primarily, an aseptic gauze sponge in a clean cavity is likely to become contaminated ultimately by the intestinal flora.

The usual tendency is for the foreign object to reach the surface, either through wound, gut or vagina. It is surprising at times how well the peritoneum can protect against these objects and hold them quiescent for weeks, months, or even years. Metal instruments seem often to cause less reaction and may remain quiescent longer than gauze.

The experimental implantation of foreign material within the peritoneal cavity has received considerable study. Various substances have been utilized in these studies, such as cork, pith, silk, cotton, linen, and other nonabsorbable material, as well as different forms of animal material which is capable of absorption. These experimental studies were carried out during the early days of abdominal surgery. Spiegelberger⁸ and Waldeyer⁸ in 1868 showed the harmlessness of aseptic ligature material in the abdomen.

Hallwachs,⁵ in 1879, by careful experimental work showed that fine, nonabsorbable sterile ligatures in the peritoneal cavity first produce a circumscribed inflammatory reaction, then become covered with a thin layer of granulation tissue and thus being isolated from surrounding structures become gradually disintegrated and removed through the agency of the tissue forces and the leukocytes.

Von Dembowski⁴ concluded from his experiments that all sutures act as foreign bodies and cause firm adhesions along their lines of inser-

tion. The clinical findings have shown that fine ligatures or sutures may be covered by new tissue within the peritoneum and remain innocuous through many years. Occasionally, however, after long periods they would excite irritation. Larger nonabsorbable sutures while usually taken care of by the tissues are more likely to cause trouble. The presence of even small bacterial contamination always increases the local reaction resulting from such material and their extrusion is by no means infrequent.

In the early days of abdominal surgery it was not unusual to find a wound in which silk was employed as a ligature in the pelvis, and which had previously healed, open and continue to discharge from a small sinus until the silk was extruded. Since the adoption of absorbable ligature and suture material such a result rarely obtains.

Foreign bodies enter the peritoneal cavity in the following ways:

1. By being placed there for experimental study.
2. By trauma, the best example being gunshot wounds, stab wounds with portion or all of the sharp instrument remaining in the peritoneal cavity.
3. Those left intentionally by the surgeon to control hemorrhage—gauze packing, clamps, catgut, silk, or linen ligatures, to aid in the completion of an intestinal anastomosis, as silk or linen ligatures, or for the purpose of drainage, as glass, rubber, or gauze.

The nonabsorbable materials left by the surgeon to control hemorrhage or to provide drainage usually are exposed to the surface and are readily removed on the third to fifth day. The absorbable sutures are permitted to remain.

4. The passage of foreign bodies into the peritoneal cavity through ulceration, as gall-stones from the gall-bladder or foreign bodies from the alimentary canal. A great variety of such objects have been reported.

George J. Heuer ⁶ reports a previously unpublished case which is of considerable interest in which peritonitis was produced as the result of a large radish within the peritoneal cavity. This patient entered the Cincinnati General Hospital, Nov. 7, 1922. About eight-thirty the same night he had inserted the large radish into his rectum for the purpose of committing suicide. His idea was to rupture the gut and bleed to death. According to the patient's description the radish was about four inches long and an inch thick at the small end and one and one half inches thick at the other. It was bent at a slight angle. He consumed about ten minutes for its insertion. Immediately afterwards the patient changed

his mind and attempted to recover the radish, using hooks and other instruments in the attempt. Having had no success he came to the Hospital for relief. He had taken one dose of salts, but had had no bowel movement.

Physical Examination.—The patient was a fairly well developed man, fifty years of age. He was lying upon his left side and complained of severe pain. General examination was negative. There was a prolapse of the mucous membrane of the rectum of about one and one half inches, which was easily reduced. Both sphincters were widely dilated and mucous membrane was seen to be much inflamed and edematous. At the very tip of the inserted index finger a rounded foreign body could



FIG. 24.—ILLUSTRATING DR. GEO. J. HEUER'S CASE OF RADISH ENTERING PERITONEUM THROUGH RECTUM, PRODUCING PERITONITIS.

be felt. There was rigidity of all abdominal muscles, but they relaxed when patient took a deep breath. Patient complained of pain over entire abdomen, but more severe over the left lower quadrant. There was no mass felt in the pelvis. The abdominal signs were clearly those of peritonitis.

On proctoscopy the patient just before operation, Heuer was impressed at once with the fact that while the lower end of the foreign body in the rectum could be brought into view, it did not occupy the lumen of the intestine; that is to say, the proctoscope passed apparently beyond the foreign body and, when a digital examination was made, there seemed to be a fold of mucous membrane between the examining finger and the radish. Further search showed a definite tear in the wall of the rectum beyond which was a white "shimmery" membrane, the

peritoneum. It appeared probable as a result of this examination that the foreign body had passed out of the rectal canal and into the abdominal cavity.

A left rectus incision was made and the peritoneum widely opened. There was immediate discharge of a large amount of purulent fluid. Intestines were intensely injected and in some places stuck together by a fresh exudate. It was evident that the patient had a general peritonitis. As soon as the fluid was evacuated and the intestines pushed aside, there appeared along the right pelvic wall an enormous red radish which the patient had introduced into his rectum. It was lying perfectly free in the peritoneal cavity. It was removed. The patient was then put in the Trendelenburg position and the intestines held up with gauze packs. The rent in the rectum was then quickly found. It was $3\frac{1}{2}$ cm. in length, lay in the midventral wall of the rectum. The tear in the rectum was closed transversely instead of longitudinally. The wound was closed after several drains were placed down to pelvis. The patient's condition continued serious and death occurred 36 hours later.

Stewart⁹ of New York exhibited to the New York Surgical Society a number of years ago a lead pencil which he had removed from the cecum of a man who entered the hospital with what was believed to be an attack of appendicitis. Careful inquiry elicited that this man while trying to relieve his constipation had passed into the rectum a lead pencil nearly six inches in length. The blunt end of the pencil was introduced first. The pencil slipped out of his fingers and was lost. The symptoms presented, which developed some time later, were pain and discomfort in the right iliac fossa. Upon opening the abdomen the pencil was felt lying in the cecum and was removed through the stump of the amputated appendix. A very interesting point to note in this case is the fact that this object was carried opposite the fecal current through the entire length of the large bowel.

Upon the passage of such objects into the peritoneal cavity peritonitis is promptly excited, producing a reaction similar to any perforative lesion. Such symptoms may be preceded in some instances by the history of surgical operation in which a Murphy button or other mechanical appliance was used. There may be a history of the patient having swallowed a nail, an open safety pin, a knife blade, a set of false teeth or other object which will cause more or less disturbance and finally become localized. Such objects gain entrance into the peritoneal cavity through ulceration of the intestinal wall. When such escape occurs the peritonitis must be treated as usual. By prompt recognition of this type of foreign body and its early removal, peritonitis can be prevented.

5. The accidental passage of instruments into the cavity during the examination of the rectum or colon, the passage of a uterine sound or in the use of the uterine curet.

There are cases on record where in the early days of proctoscopy, during the examination the proctoscope was passed through the intestinal wall. The surgeon in one case recognized the accident, promptly opened the abdomen, closed the rent, and saved the patient. With care such an accident may be avoided.

The occurrence of puncture through the uterus from instrumentation is more frequent. It may occur from efforts of the patient to produce abortion, from the professional abortionist's efforts to empty the uterus, and in a few instances from the efforts upon the part of surgeons to remove detritus from the uterus. Because of the attitude of the profession toward curettage and the care that is advised in the use of this instrument, this accident is rarely seen at present.

In some instances perforation of the uterine wall gives rise to little immediate disturbance, while in others the reaction is prompt and severe. The peritonitis excited may be quite severe or somewhat mild in type. The indication for immediate section is plain in such cases.

6. By their accidental implantation by the surgeon or his failure to remove them during surgical operative procedures.

Foreign bodies accidentally left in the abdomen during surgical operations, or during the treatment following, are embarrassing occurrences.

The profession has very generally recognized the possibility of such an accident. As early as 1901, August Schachner¹⁰ reported a case in which this accident occurred, and collected 46 cases which added to Neugebauer's⁷ collection of 109 cases made 155 cases in the literature up to that time. C. White¹¹ reports 2 cases in which he removed objects not placed in the cavity by himself.

Crossen⁸ from 1888 to 1907 collected 50 cases from the literature. Among the objects left were forty-one forceps, three drainage tubes, two finger rings, one Nélaton catheter, one glass irrigator, one scissors, one piece of an instrument, and one pair of spectacles.

Schachner's analysis of Neugebauer's report shows that of the 109 cases reported 43 resulted in death, which does not mean that the presence of the foreign body necessarily caused the death of the patient. In 19 cases which recovered the foreign body was a sponge. In 64 occasions a gauze sponge, napkin, or mull compress was left.

Nature unaided successfully dealt in different cases with four drainage tubes, one Richelot clamp, nineteen artery forceps, one seal ring, one glass fragment from a burst irrigator.

From a surgical standpoint the greatest amount of importance attaches to those left by accident during surgical operation. All surgeons know how easy it is to leave a sponge, a piece of gauze or even a surgical instrument in an abdominal wound, and how difficult it is to find a sponge in some cases even where the count has been properly checked, and one must certainly be left. They also recognize the fact that in the search for a missing sponge two important things must be borne in mind: first, the human equation, by which is meant the possibility of error in the count when the sponges were put up in packages, and again when the check of sponges was made after the operation; second, the resistance of the patient must be considered in cases where the check of the number of sponges or instruments is made and search is instituted. This search may not be carried beyond the point where the safety of the patient is likely to be jeopardized.

Whenever there has been an unsatisfactory sponge count the surgeon feels uneasy a long time, but in the absence of symptoms or evidence of the presence of a sponge he becomes confirmed in his opinion that there was none left.

Sometimes when the suspicion of this accident is strong, or when there has been no suspicion or even suggestion of it, there may be noticed a persistent hardness near the abdominal wound or in the pelvis, with the persistence of a discharge of serum from an otherwise clean wound or of a suppurative discharge from an infected wound or through the vagina in a vaginal section. Accompanying this there may be complaint from the patient of pain and tenderness over the indurated spot. Again there may be constipation, difficult and painful passages from the rectum, free seropurulent discharges from vagina, rectum, or bladder. In some cases that were reported pus and blood were escaping from the bladder with dysuria.

With any of these symptoms occurring after abdominal or vaginal section, the surgeon should give the patient immediate attention. A careful study will disclose the cause. In handling such a case a general anesthetic is necessary as a rule. The foreign body can usually be found in the center of the indurated mass and removed.

H. S. Crossen³ presents a very unique method of preventing the accident of leaving sponges in the abdominal cavity. This consists in the employment of a gauze bag to which the sponge is firmly attached by one end, the other going into the abdominal cavity. The bag is left

attached to the towels outside the wound and is removed with the sponge when no longer needed.

Perhaps the attachment to the gauze of metal rings which lie outside of the abdomen makes the most satisfactory method, and if a ring is carried into the abdomen with a sponge and left there its presence can be readily detected by a skiagram.

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CHAPTER II

WOUNDS AND WOUND TREATMENT

Wounds of the abdomen in many respects differ materially from wounds in other portions of the body. These wounds are open or closed. Under open wounds, incised or stab wounds occur with considerable frequency. Contused, lacerated, and punctured wounds are met somewhat infrequently. Infected wounds are seen but seldom, because of the amount of protection afforded this portion of the body by its position and the clothing. Gunshot wounds are relatively frequent.

The gravity of most abdominal wounds depends largely upon the amount of damage done to the viscera, particularly the hollow organs. In this they are somewhat different from wounds in other portions of the body.

STAB WOUNDS

This class of injuries results from the thrust of knife, sword, bayonet, or other sharp object. They vary greatly in extent and in the amount of damage done both to the abdominal wall and to the viscera. Very largely this damage depends upon the manner in which the violence is applied.

The wounds produced by the stiletto while apparently trivial may be very grave. The thrust of a rapier, a saber, or that of a bayonet may make a frightful wound. But the appearance of the external wound should not be taken as an index of the internal injury.

Again, a wound of the neck in a transverse direction may seem a horrible affair when the head is thrown backward. When the neck is flexed it may seem a minor affair. The position of the limb, either in flexion or extension, materially changes the amount of gaping.

Modern methods of warfare have so changed that the percentage of such injuries coming under observation is almost negligible.

This class of wounds presents the following local symptoms: pain, hemorrhage, and retraction of tissue or gaping. The latter symptom varies greatly with the extent and direction of the wound.

The first evidence of an incised wound is a sharp stinging pain which is frequently overlooked during the excitement of combat. Following

sharply upon the pain occurs hemorrhage which may be concealed or external. If the bleeding occurs externally it gives prompt evidence of injury. Internal hemorrhage gives quite characteristic signs of its occurrence, but they may not be evidenced until some time has elapsed.

Hemorrhage is in part the cause of some of the constitutional symptoms of an incised wound. Thirst, dyspnea, pallor, faintness, and syncope are the systemic effects of hemorrhage. In case the amount of hemorrhage is large, the patient may immediately become unconscious and there is marked evidence of shock.

This is shown by extreme pallor, lethargy, rapid pulse, cold, moist, clammy skin, subnormal temperature. Such patients may not go into unconsciousness, but this is frequently observed. This train of symptoms may persist until death.

The evidences of recuperation from shock are returning warmth to the surface, increase in the volume of the pulse, the restoration of color to the skin, rise of temperature to normal and the restoration of consciousness.

Every stab wound of the abdominal wall is potentially a penetrating wound and also possibly a perforating one as well.

Superficial Stab Wounds.—These are treated in identically the same manner as incised wounds in any portion of the body. The indications for treatment are the control of hemorrhage, the removal of foreign bodies, including bacteria, and the proper coaptation of the wound surfaces. These steps should be carried out with painstaking attention to detail.

The wound should remain without interference until complete asepsis may be applied, except in cases where the hemorrhage is serious. Under such circumstances it may be necessary to disregard asepsis to save life. Such a contingency but rarely presents.

The control of hemorrhage may be accomplished temporarily or permanently. The temporary measures for the arrest of hemorrhage which are accessible are compression, acupressure and forcipressure. The permanent means of arrest are torsion and ligation.

The methods of compression in use in abdominal injuries are digital or mechanical.

Digital compression over a bleeding vessel controls the bleeding completely, but may be used only for a short time. Compression by means of a gauze pad or towel may be effective. Heat, cold, and chemicals are too uncertain to be dependable in this class of injuries.

Compression by forceps gives complete control and may be used both for temporary and permanent control of hemorrhage.

The use of torsion or the ligature is the method usually employed for permanent arrest of hemorrhage. The field about a wound should be thoroughly cleansed with benzin or alcohol, and after drying thoroughly, tincture of iodin ($3\frac{1}{2}$ per cent) may be employed for sterilization. The wound surfaces may also be painted with iodin. Thorough removal of foreign substances including clots is then accomplished. The wounded surfaces are then brought into apposition, each layer being carefully approximated so that there will be small tendency to leave a weak wall.

When a nerve of considerable size is cut it should be searched for and the ends approximated. With but few exceptions such wounds should be closed without drainage.

All persons suffering from accidental wounds of the abdomen should receive an injection of 1500 units of antitetanus serum (particularly true of war wounds).

Penetrating Stab Wounds.—Every incised wound which is found to penetrate the abdomen is potentially a wound of one of the viscera.

All wounds of this character occurring in civil life or during warfare should be explored sufficiently to determine the presence and extent of any intestinal injury, also the presence of any bleeding vessel. The only possible reason for delay is the belief that the steps necessary for such treatment will produce fatal shock.

If the wound does not involve one of the viscera the small increase in its size for thorough investigation of the abdomen adds but little to its gravity. If an injury to a viscus or to a large vessel is present, the investigation will probably save the life of the individual when death would likely result from noninterference. The presence of pain, general abdominal tenderness, rigidity, and other evidence of visceral injury or the evidence of serious hemorrhage call for prompt abdominal section.

Measures to overcome shock may be necessary before such procedure may be undertaken. It is well to remember that patients rally more quickly from shock when bleeding vessels are closed.

Heat to the surface, heated saline solution in the rectum or under the skin, the internal administration of ammonia, whisky, or brandy, and the use of cardiac stimulants hypodermically will aid recovery from shock.

Visceral wounds occurring from stab wounds will probably be less numerous than in gunshot wounds. They usually bleed more freely than gunshot wounds. This is particularly true in the solid viscera. Hemorrhage from wounds of the liver and spleen may be controlled

temporarily by pressure. Because of the friability of such tissue suturing must be done carefully and it may be necessary to suture over gauze in some cases to get the sutures to hold.

Intestinal wounds after control of hemorrhage are to be closed without constricting the lumen. Rarely the intestine is entirely severed in several loops. A resection of the intestine may be necessary under such circumstances.

Lacerated and contused wounds are to be cleansed in the same way as incised. When considerable tissue defects are present and large dead spaces remain immediate closure may not be indicated and dependence may be placed on drainage with secondary closure. Otherwise general surgical principles are to be employed for these and for punctured wounds as well.

No treatise upon wound treatment is complete which fails to mention the danger from contamination with the bacillus of tetanus. This organism, which was discovered by Nicolaier, 1880, and isolated in pure culture by Kitasato in 1881, is the active cause of tetanus, an ever present menace in all accidental wounds, particularly those occurring in warfare.

The mortality in previous wars from this infection was very high, while in the American Army in the World War it was extremely low. This result obtained because every wounded soldier received 1500 units of antitetanic serum.

In the treatment of gunshot wounds in particular, therefore, and in fact all other wounds the prophylactic dose of 1500 units of antitetanic serum should be employed. This rule should obtain both in military and civil practice.

GUNSHOT WOUNDS

The term gunshot wound is used to cover any injury resulting from discharge of gun powder or any high explosive. This includes injuries resulting from either primary or secondary missiles or from pieces of the firearm itself in case of explosion. A primary missile is one propelled directly by the force of the explosion. A secondary missile is one set in motion directly or indirectly by a primary missile. The writer has personally seen the button upon the neck band of a soldier broken into two fragments and these fragments, in company with the bullet separated from its jacket, making four pieces in all, driven into the mediastinum. The fragments were removed from their location three inches below the suprasternal notch through the wound of entrance, which was in the neck just above the manubrium.

Gunshot wounds are often very innocent looking because of the apparently small amount of damage done at the point of impact, when, in fact, such an injury is of the most serious nature. The damage done by a missile cannot with accuracy be estimated by the appearance of the wound of the skin. Very small fragments of steel, almost a shaving, causing only a slit in the skin often produce the most extensive destruction of the soft parts.

The fragment seems to take on a movement similar to that imparted to a billiard ball by "English" and while for the most part retaining its course, it does considerable tangential damage.

Gunshot wounds depend for their outcome upon the character of the missile, its velocity, the rifling of the weapon, the distance from the point of impact to the point of discharge; but more particularly upon the amount of hemorrhage resulting and upon the presence of an injury of one of the hollow viscera of the abdomen. The presence or absence of infection is always an important factor in the outcome.

There is a marked difference between the wounds by the missiles from the small arms in use in civil life and those resulting from the high power, small calibered rifle employed by most armies. The larger number of wounds in civilian communities occur as a result of the use of a pistol, varying from .22 to .45 caliber, with a shell charged with black powder having a maximum velocity of 700 feet per second. Most of these wounds occur at close range, and powder burns are frequently seen. They usually happen during a drinking and eating bout when the stomach and intestines are distended with food and when peristalsis is marked. Therefore, they are at once more serious than war wounds from rifle or machine gun fire with the possible exception of cases in which the missile has struck one of the pelvic bones, the spine or a rib. The high-powered rifle may cause extensive destruction of tissue when any bony structure is hit either within the first 800 yards of its flight or over 1300 yards. The damage is much less when the bone is struck at between 800 and 1300 yards, because the flight of the bullet is more steady at this distance, and it makes a perfectly round hole, since the so-called explosive force due to its irregular rotation on its horizontal axis in the first and last portion of its trajectory is lost when its flight is steady. Even then there is sometimes considerable bone destruction in the middle portion of its course.

Sometimes an army bullet carries into the body of the wounded man a fragment of clothing, a button, or other substance. On one occasion during the World War a soldier came into my service who was wounded while carrying in the pocket of his blouse a .30 caliber bullet. This was

struck by a rifle bullet causing it to explode. Both bullets and the shell of the last entered his body.

There is such great variety of firearms that any attempt to describe them would go beyond the scope of this monograph. From the toy pistol to the highest powered naval guns there is room for wide variation.

The missiles vary just as greatly. Among these may be mentioned missiles varying from the smallest bird-shot to buck-shot weighing 150 grains, the .22 caliber pistol bullet to the massive shell of the artillery. Of a necessity such diversity of missiles provides the greatest possible variety in the character of wounds. The high explosive shell at present results in extensive tissue destruction. Certain shells carry poisonous gases which also add to the casualties in war.

Ordinarily a slow moving object causes less damage to the tissues at the point of impact than one with great velocity. Under certain conditions this is not true of bullet wounds. The rifle ball traveling at the rate of 2500 to 3000 feet per second makes a formidable missile. The tissue destruction of such a bullet in full flight may not be extensive. It has great stopping power, however. A bullet moving more slowly with marked rotation may cause more tissue destruction. Ricochet is the term applied to the movement of a bullet when it is deflected from its true course. Under such condition the wounds resulting may be severe. This bullet is often deformed and also carries in addition to the force of gravity a rotary and tumbling motion.

Upon the rifling of the barrel of the gun depends the steadiness of the flight of the projectile throughout the major portion of its course. This imparts the rotary motion to the ball which produces at times the so-called explosive action of a bullet.

The distance from the point of discharge to that of impact plays a very important part in the effects of the missile. Within from two to six feet the powder markings of a wound produced by black powder may be considerable. Smokeless powder may not leave such markings.

Shotgun wounds at close range are particularly destructive, while at some distance, because of scattering of the shot, the resulting wound may be trivial.

More important still in determining the outcome of a given wound is the particular tissue which is injured. The skin is elastic, stretches considerably, and is well supported at the point of entrance by the tissue beneath. The wound of entrance is different in these injuries, the rifle bullet producing a small round opening or a slit in the skin, while the wound of exit may be extensively torn because the skin at the point of exit

has no support. In case a bone has been injured there is even greater damage if fragments are carried through. In case one or more secondary projectiles accompany the primary missile and remain in the tissues the wound of entrance is much larger. There is considerable contusion around the wound of entrance of a rifle bullet, which may result in a slough in a few days and the size become equal to that of the wound of exit.

The wound of entrance from ordinary pistol bullets is usually much larger at first than the wound of exit, unless some fragments are carried with the bullet as it escapes.

Gunshot wounds of the abdomen are among the most serious injuries whether occurring in civil life or during warfare. For purposes of study they may be divided into nonpenetrating, penetrating, and perforating wounds. Nonpenetrating are never as important as penetrating wounds. The latter are never as serious as those which perforate the viscera. The amount of damage done within the abdomen is usually less with the small high-powered bullet than with the larger sized pistol ball. Wounds of the large nerves cause loss of function in the region they supply. Injury to the nerves supplying the abdominal wall are also likely to result in hernia.

Wounds of the larger blood-vessels by either form of firearm are likely to prove promptly fatal, depending entirely upon the amount of hemorrhage.

Wounds of the smaller vessels are evidenced by shock, syncope, collapse, and considerable intra-abdominal pain.

In civil life there is considerable tissue destruction from wounds caused either by pistol balls of large caliber or from shotgun wounds at short range. The former damage the tissues of the wall considerably and lodge there or pass through, making a perforating wound of the abdominal wall but not necessarily of the peritoneum or of the viscera.

Superficial Gunshot Wounds.—The nonpenetrating war wounds from rifle or machine gun fire are usually not of serious nature. The particular dangers of such injuries are hemorrhage, shock, and infection. The hemorrhage from such wounds depends upon the location and size of the vessel injured and the promptness with which surgical aid is obtained.

Shock in this class of injuries depends upon the conditions under which the wound was received. Such a wound occurring after a fatiguing march or a protracted battle with prolonged exposure to inclement weather makes for shock. Hemorrhage adds very greatly to the amount of shock.

The danger from infection in these nonpenetrating injuries is much

greater during warfare than during civil life. It was found by surgeons in the late war that nearly every wound showed bacterial infection when brought in and a very large percentage showed the presence of the *Bacillus aerogenes capsulatus* of Welch. Prompt surgical care of such cases should bring them through without great mortality.

Wounds in which the projectile passes through the abdominal wall without injuring a viscus are less serious than those in which the missile remains. If clothing or other foreign material is carried into the wound the chance of infection is enhanced.

Shotgun wounds of the abdomen at close range are very serious accidents. The entire wall of the abdomen may be torn away and marked damage done to the viscera.

On the other hand, shrapnel wounds are just as serious to the parietes of the abdomen and in many cases more so.

These wounds of the abdominal wall are recognized by the local symptoms and by the conditions under which they are produced. The symptoms are similar to wounds of the same nature elsewhere. These are hemorrhage, gaping, pain, loss of function, shock. The hemorrhage is for the most part external and is easily recognized and controlled. There may be considerable shock, which must be counteracted at once, as soon as the hemorrhage is controlled. The damage to the soft parts is so marked that considerable dissection may be necessary to purify the wound and bring the edges into apposition. When these steps are carried out promptly the outcome of this particular class of cases is assured.

In war wounds the presence of infection, particularly with gas bacillus, necessitates a complete débridement and purification of the wound to obtain primary healing. Such extensive procedures are only occasionally necessary in civil life. The pistol wounds, particularly, heal kindly. Perhaps only in the immediate vicinity of the entrance is there any contamination.

Some years ago I was forced to the conclusion that the most important consideration in treatment was proper purification of the wound. In many cases simple cleanliness is all that is necessary. When a chemical agent is employed certain properties are requisite. These are bactericidal action and the very smallest amount of destructive effect upon tissue. Of all the chemicals which are used in wound treatment nothing equals alcohol in proper dilution. Sixty per cent strength will usually prove the most satisfactory. In the surgery of bones a weaker dilution is preferable, taking into consideration the preservation of the osteogenetic function.

The use of iodine is permissible in badly infected wounds and for

sterilization of the field at operative procedure. Subsequently its use will cause necrosis and inhibit cell proliferation.

Dakin's solution employed while fresh and properly prepared seems to act well in some cases. Its chief value seems to lie in its cleansing property since it digests the necrotic tissue rapidly and facilitates drainage. It must be borne in mind that when employed in the neighborhood of large vessels which have been damaged, it liquefies the clots and the animal ligatures. This sometimes results in fatal hemorrhage. The normal tissue resistance must not be overlooked in handling wounds.

Drainage is to be used freely in infected wounds.

After a wound is properly cleansed and dressed, it should remain at rest.

Pressure support to the tissues bringing the raw surfaces into apposition is most valuable.

Penetrating Gunshot Wounds.—Penetrating wounds of the abdomen occurring in civil and military life in the large majority of cases are perforating. For this reason and because of the extreme gravity of such perforations the immediate performance of abdominal section is indicated. The only possible reasons for noninterference are positive evidence that such perforation has not occurred and the fact that the patient's condition will not justify it.

The presence of general abdominal pain, tenderness; and muscular rigidity point to the occurrence of perforation. To determine this point an exploration of the wound by open dissection may be necessary. If this is done proper preparation should be made for handling any visceral injury.

Theoretically the same rule should obtain in all perforating wounds during war. Practically this is not correct.

The wounds produced by small calibered rifles may pass through an intestine and the patient go to recovery. This is explained by the small opening, by the empty canal and by prompt agglutination. Many of these cases, therefore, do better when kept at rest with heat applied to the surface and when given hot water and the usual measures for the treatment of shock.

Of all measures for the control of shock nothing equals heat. In addition the use of atropin sulphate and morphin sulphate will prove valuable. Unless the injury involves the stomach itself no harm can come from the ingestion of hot brandy. The subcutaneous instillation of normal saline solution to fill up the vessels is beneficial. The addition of adrenalin chlorid solution may tide over the emergency.

Moribund cases cannot be saved. Those which show some recupera-

tive power but show evidence of hemorrhage should come to operation promptly. After hemorrhage is controlled the regeneration of blood is rapid and may be aided judiciously.

Abdominal section for perforating gunshot wounds should be conducted systematically and with the utmost celerity, giving due consideration to care in each step. A few moments of wasted time may turn the balance against recovery.

The abdominal incision should be sufficiently free to permit the rapid exploration of the organs without evisceration. All hemorrhage should be promptly controlled. Hemorrhage from the liver may be instantly controlled by grasping the lesser omentum with the finger in the foramen of Winslow. Pressure upon the splenic artery and vein fairly well controls hemorrhage from a wound of the spleen. The group of vessels at the greater curvature of the stomach-inosculates freely with the vessels of the spleen. This point should be borne in mind. Large gauze packs are useful in controlling deep peritoneal hemorrhage. Wounds of the superior mesenteric artery are likely to prove fatal before the surgeon sees the patient. Quick work is essential. After hemorrhage is controlled the intestines are systematically examined so that no wound is overlooked. It is just as fatal to overlook the last wound of a dozen or more as to overlook them all. Before closing any intestinal wounds, all should be found. Then the decision may be properly made as to the necessity of intestinal resection. In many cases this is the quickest and best way to complete the repair promptly. Careful attention in all intestinal suturing must be given to the blood supply at the line of suture. This is particularly true to prevent leaks from necrosis. When suturing single wounds of the intestine the line of repair is to be placed so that no constriction of the lumen will result. The lines of suture may be tested by forcing gas against them and the finger may test the patency of a resection. These steps are unnecessary in times of stress.

The surgeon must determine whether there has been too much peritoneal soiling to do without drainage. He should remember that in battle this patient is to pass to the care of others and be governed accordingly. Closure of the abdominal wound should be carefully made to prevent hernia. All these patients do better after operation if the stomach is washed out. This does away with nausea, hiccough, and vomiting. The measures for the treatment for shock must be kept up vigorously. The vessels may be filled with fluid after hemorrhage is checked with perfect safety. Transfusion is indicated in cases of severe hemorrhage.

The postoperative treatment otherwise does not differ from any operation of gravity. These patients may take water even in gastric wounds

at the end of thirty-six to forty-eight hours. In seventy-two hours liquid food may be administered.

RUPTURE OF THE INTESTINE

Traumatic rupture of the intestine without external evidence of injury is by no means infrequent. This condition is caused by sudden sharp blows on the abdomen by blunt objects, as the end of a piece of timber, the impact of a sledge hammer, by compression from the passage of the wheel of an automobile or a wagon over the abdomen, from compression of dirt or rock or a crushing external force. It may also result from the sudden increase of pressure within the intestine. Cases are recorded where men working with compressed air have turned the hose into the rectum and caused this injury. Rupture of the intestine has also been caused by rectal instrumentation. John Mason Williams reported a case in which he was using the proctoscope and discovered that it had passed into the abdominal cavity.

Rupture of the intestine is not an uncommon accident in coal mines and other excavation work. A case of rupture of the duodenum from such compression came under the observation of the writer.

These injuries for the most part occur as the result of the impingement of the causative object pressing the intestine suddenly against the promontory of the sacrum, against the vertebral column, or against the pubic arch or ilium.

In one case the writer observed this injury from a workman in a shipbuilding establishment permitting a large hammer to slip, striking him a severe blow just above the pubic bone. In this case there was no ecchymosis, swelling or other external evidence of local injury.

There is a wide variation in the extent of the injury in this class of cases. From a simple pinch of the gut with a localized ecchymosis to the complete rupture and wide laceration of the intestine. In some instances, particularly when the blow is over a portion of the intestine closely attached to the bony structures, as the junction of the sigmoid and rectum, there may be a complete tear through the entire structure of the gut.

According to Lockwood¹ as a rule the small bowel is completely, while the large bowel is only partially, severed in such injuries. My own experience has not been in accord with this.

Undoubtedly the most important factors outside of the force of impact in determining the character and extent of the injury are the distention of the gut and its fixation. The greater the distention the more

firm its fixation and the closer the impact to the fixed portion the greater the extent of laceration. A mobile bowel pinched against the pubes where the impact is against only a portion of the lumen, as occurred in one of my cases, makes but a small rent.

One can readily understand how a large, square end of timber pushing suddenly and with great force against the promontory of the sacrum, could cause a complete section of an intestinal loop, or in some instances more than one loop. This fact must be borne in mind in this type of case and also in cases where sudden entrance of compressed air into

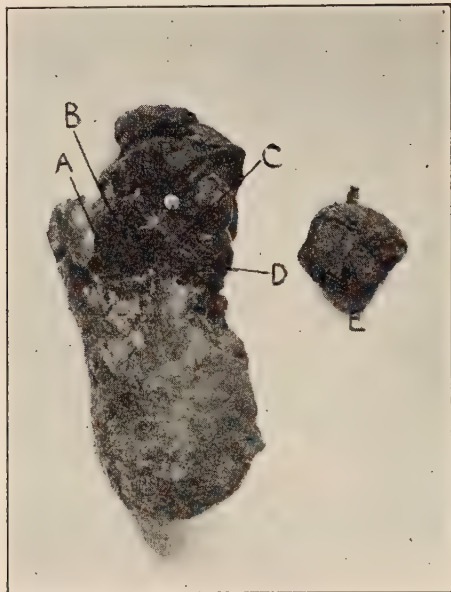


FIG. 25.—RUPTURE OF APPENDIX FROM TRAUMATISM, ENTEROLITH ESCAPING.
(Case of Dr. James S. Chenoweth.)

the gut is the causative factor. Injuries from this source may show multiple lacerations, often with considerable hemorrhage and great shock.

The portions of the intestine which are not covered with peritoneum may be ruptured extraperitoneally and this possibility must be considered in their management.

Lockwood¹ collected 652 cases of traumatic rupture from the literature occurring in civil life. The site of the rupture was in the small intestine in 90 per cent, while only 10 per cent of the cases affected the large gut. The cecum, transverse colon, and pelvic colon were affected in the order named. A few cases of traumatic rupture of the appendix are on record. Dr. Chenoweth reported one such case.

Diagnosis.—The symptoms in these accidents differ greatly in degree, owing to the great difference in the causative force and the site and extent of the injury. In many cases the symptoms are not prompt in development and are frequently not so severe during the first few hours after the injury as the extent of injury would seem capable of producing. The symptoms are usually more pronounced and develop earlier after the injury when the intestine is full. There seems to be a temporary paresis of the gut immediately after the injury which prevents peristalsis for a time and also benumbs the sensory apparatus of the damaged loop. The pain sensation is not marked in the small bowel, which in part accounts for the slow reaction. When the intestine is empty and there is little escape from it, the peritoneum does not resent the injury at once, but if the bacterial flora in the bowel is abundant and virulent, the onset of pain is prompt and is proportionate to the response of the peritoneum to the presence of the contamination. In some of the minor injuries the peritonitis excited is localized, and the reaction is localized as well. Pain at the site of injury, local rigidity and tenderness occur, with increasing induration in the abdominal wall. This course is likely to follow extraperitoneal rupture at first, but later the symptoms of peritonitis develop, perhaps as late as three or four days.

In other cases there may be but little pain at first, but in twelve or fourteen hours local soreness and pain are noticed and there may be a rapid progression of symptoms indicative of peritonitis. The temperature may be subnormal at first, and the pulse compressible in the small injuries, rapid and feeble in the more severe. When these symptoms in a short time change and the temperature rises, the pulse becomes fast and wiry, angry in type, localized and general tenderness marked, and the entire abdomen rigid, the diagnosis of rupture of the intestine with peritonitis is justified. If the patient is to receive any benefit, this is the time for intervention. Vomiting is an important symptom, occurring usually soon after the injury, but may subside when the stomach is empty, to appear again when peritonitis sets in. Lockwood states that it occurs early if the lesion is high in the small bowel. The abdominal tenderness is progressive. Distention may not be present early when the lesion is in the upper part of the small intestine, and in the early hours the abdomen may be scaphoid in type. As peritonitis progresses and a paralytic ileus develops, the distention is very decided. When the intestinal gases escape into the peritoneum, there is absence of liver dullness, a very valuable sign. The presence of gas in the peritoneal cavity may be shown in the skiagram, if under sufficient pressure. If so, the viscera can be outlined and the diagnosis made positive. In some cases

surgical emphysema is present, particularly in extraperitoneal injury of the colon.

Prompt recognition of this condition is of the greatest importance. If an error be made, it is much safer for the patient if he is opened when no lesion presents than to delay when one is present. In the first instance recovery is practically sure and early operation saves these individuals while delay seals their doom. This field of surgery demands boldness and promptness. Lockwood says that practically all cases of rupture of the bowel are fatal if not operated upon. He operates upon all persons who, following a blow upon the abdomen, a crushing injury, or a fall, complain of severe abdominal pain which lasts for more than four to six hours, and is associated with tenderness, vomiting, rigidity, and an increasing pulse rate.

Prognosis.—The prognosis is very grave. Those with no operation die. Those who are operated upon late show a very high mortality. Those operated upon within eight hours have a chance. Lockwood from 1914 to 1918 saw 27 cases. Fourteen came too late for operation. All died. Of 13 operated cases, 5 recovered. Rupture of the bladder and retroperitoneal injuries with kidney lesions complicated two fatal cases.

Treatment.—In case of severe shock following such an injury, with abdominal tenderness, the abdomen should be opened without delay, measures to overcome shock having been instituted. It should be borne in mind that great shock means either hemorrhage or a great disaster. If hemorrhage, the blood loss must be checked. Normal saline may be given intravenously when ready to open the abdomen. Heat to the surface, morphin and atropin, with a quick cardiac stimulant, as amonia or whisky, will bring about reaction. Great and persistent pallor and thirst indicate hemorrhage. If the symptoms are very mild, one may be led to think the injury superficial. Such a patient should go at once to a hospital. If there is absence of liver dulness, operation is indicated. If his condition warrants the use of the x-ray, it may help in diagnosis. When there is doubt, if the symptoms are increasing, operation is indicated provided the condition of the patient permits.

The incision should be wide and made according to the site of the blow, usually in the median line. Hemorrhage must be controlled at once and all openings in the intestine closed quickly, but securely. Rapid peritoneal toilet with immediate closure should be made if the operation is within four hours and there is not much soiling; with later operation drainage is likely to be necessary. In the extreme cases, open the wall under local anesthesia, place the drains quickly and get out. When

the wounded gut presents and the patient's condition does not justify protracted effort to repair the leak, either bring the injured loop into the wound or place a drain in it. When the duodenum is injured and the condition grave, all that may be done safely may be to place a drain in contact with the torn portion. These measures are only for cases *in extremis*. Wherever possible the leak should be repaired. I look upon duodenal injuries as the most grave, because of the difficulty of approach and the time consumed in the necessary manipulation. In retroperitoneal wounds of the colon, approach may be made by severing the outer peritoneal attachment, or the abdominal wall may be opened extraperitoneally to reach the rupture.

Multiple ruptures occur in 20 per cent.¹ When conditions justify operation, all ruptures should be searched for and repaired. The other intra-abdominal organs should be examined quickly and the entire operative procedure completed with celerity.

The patient should receive one pint of normal saline solution by rectum during the first three hours; should be kept warm and given stimulants with morphin (gr. $\frac{1}{4}$) to prevent restlessness and relieve pain. Gastric lavage at once will prevent nausea. It should be repeated if necessary.

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CHAPTER III

EXAMINATION OF THE ABDOMEN

PHYSICAL EXAMINATION

In the examination of the abdomen there are a number of things to be borne in mind which may lead to the avoidance of error.

The first consideration is to obtain the confidence of the patient that no unnecessary pain or discomfort is going to be caused. If the attendant comes in with cold hands, he should either wash them in warm water or after the use of cold water should rub them until thoroughly warm. The abdomen becomes immediately rigid on contact with anything cold. The approach should not be abrupt, the manner should be cordial. A few minutes of conversation is well spent. In the examination itself, I have been impressed with the increased amount of information which may be obtained from the flaccid hand placed flat upon the abdomen. The more gentle the pressure the more knowledge that can be obtained of the structures beneath. If the patient's attention is distracted during the examination the relaxation is increased and the observer may tell from the facial expression just how much pain the patient suffers. After this the examination by finger tip pressure is more readily made, percussion and auscultation may be completed without objection on the part of the patient. The facts so elicited are very important.

Masses may be detected within the abdomen. The capping of omentum or intestines may be made out by palpation. The dulness resulting from localized abscess is made out by percussion. A distended bladder or loaded intestine may be detected in the same way. Percussion over the liver showing absence of liver dulness indicates a perforative lesion.

The presence of free fluid may be detected by the percussion waves imparted. Changing locations of dulness also aid in its determination. Rarely succussion will elicit it when the amount is large. A general anesthetic greatly facilitates the detection of intra-abdominal masses.

No abdominal examination is complete without a vaginal and rectal examination.

PNEUMOPERITONEUM.—The use of air, oxygen, or carbon dioxide injected into the peritoneal cavity for diagnostic purposes with x-ray guidance is one of the recent advances in surgery.

The injection of air into the peritoneal sac for diagnosis was first practiced in 1902, by Kelling⁷ by means of the cystoscope. In 1903 its use within the abdomen to overcome negative intra-abdominal pressure and for the prevention of recurrent adhesions was adopted by Bainbridge,¹ of New York. It was not until 1910 when Jacobaeus,⁸ of Stockholm, adopted the method in the direct observation of the pleural and peritoneal sac by direct thoracoscopy and laparoscopy.

It was but a short time after the initial work of these two men that Weber,²⁰ 1912, showed that many of the intra-abdominal viscera could be visualized by this method. Lorey's⁸ work in the same year in a case of ascites is worthy of mention. Following this pioneer work the method came into more general use.

Rautenberg¹² was one of the earliest workers in this field to undertake systematic observations by this method. His early observations were confined to cases of ascites (1913). Later (1914) he presented his work before the Congress of Clinical Medicine at Wiesbaden. He emphasizes the necessity of an empty intestinal tract and bladder for successful observations. In his early cases he used a pneumothorax apparatus. Later he employed an ordinary bellows pump and an aspirating needle. The earlier observations were confined to the structures in the upper part of the abdomen, the spleen, the gall-bladder, and the liver, particularly. Subsequently the scope of the methods has been considerably enlarged.

The impression is gained from Goetze's⁵ article, in 1918, that he evolved this method, totally independent of previous observations.

Tierney,¹⁵ of St. Louis, when this work was first undertaken was under the impression that he was entering a new field and was surprised to learn of the work which had been done previously.

Luckett and Stewart,⁹ and later Dandy,² were the first to employ pneumodiagnostic methods in the country. Their work at this time was confined to the cranium.

Stein and Stewart,¹⁴ in July, 1919, review the literature and describe their technic.

Dandy,² in September, 1919, calls attention to the value of Roentgen diagnosis in perforations of the alimentary canal, and selects hydrogen gas because of its low molecular weight as the best substance for use in this method.

Rosenblatt,¹³ in 1919, accidentally produced a pneumoperitoneum in the course of an attempted artificial pneumothorax and concludes it is innocuous.

Emerson³ concluded from his study that excessive intra-abdominal

pressure artificially produced caused death in animals from cardiac failure before the pressure produced a marked asphyxia.

Orndoff,¹⁰ of Chicago, in 1919, reports over 100 cases of induced pneumoperitoneum using air, oxygen, and carbon dioxid.

There seems to be but little choice between the three substances. Carbonic dioxid is absorbed more rapidly and causes less discomfort. If the observation will be prolonged, oxygen is better.

The scope of investigation by this method has been considerably enlarged since its first employment. In the Louisville City Hospital it was first employed by Dr. Ellars. Subsequently a considerable number of cases have been examined with this method by H. H. Turner,¹⁷ to whom I am indebted for the material which he has placed at my disposal. The work has been done in the x-ray laboratory, and the patients have been selected for examination from the surgical, the gynecological and obstetrical services conducted by the medical department of the University of Louisville.

It has been found that the method is comparatively safe if the amount is kept within proper limits, so that there is no interference with respiratory or cardiac function.

The method of injection employed for the most part is simple. An ordinary tank of oxygen or of carbon dioxid is attached by a rubber tube to a bottle half filled with water, through which the gas passes, escaping through another rubber tube connected with a manometer. The latter, in turn, is attached to a rubber tube at the end of which is attached a long aspirating needle.

The manometer registers the amount of pressure of the gas and enables the operator to keep the pressure below the danger point.

The technic employed consists in painting the entire abdomen with iodine, placing the patient supine on the table, then after selecting the point of puncture, this point is again painted. Then the wall at this point is cocaineized. In addition to infiltrating the skin with cocaine or novocain, the anesthetic is usually injected deeply into the muscular planes and also into the peritoneum. When the anesthesia is satisfactory, the puncture of the wall is made with the sterile needle. The resistance of the abdominal wall will indicate when the needle enters the peritoneal sac. When the operator thinks the needle is in the cavity, the gas is permitted to pass slowly through it. If the needle has not entered the cavity, the manometer at once registers a rapid increase in pressure. By moving the needle, it will be found that after entering the cavity, the pressure in the instrument will fall rapidly and remain around zero. One to two liters of oxygen or other gas is the amount usually used. Larger amounts

are employed for the abdominal examinations than are used in outlining the pelvic organs.

Peterson,¹¹ of University of Michigan, has reported some 325 cases in which pneumoperitoneum has been employed in his clinic. He has injected the gas through the uterus for two reasons, one, to get into the cavity, the other to determine the patency of the fallopian tubes. He

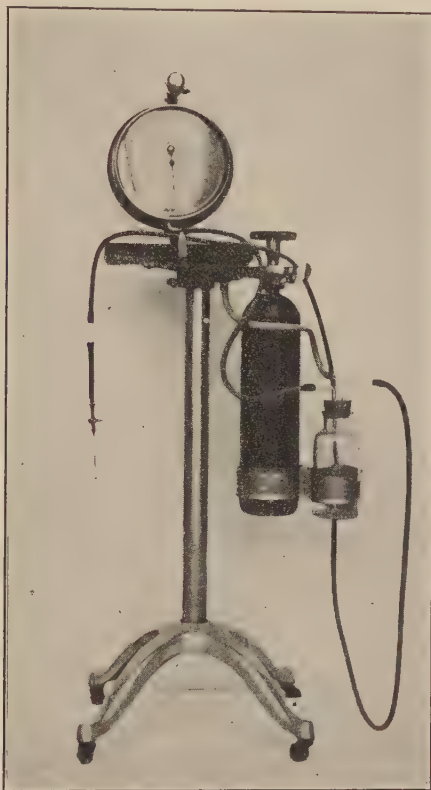


FIG. 26.—APPARATUS FOR INJECTING AIR. (After Turner.)

Tycos manometer. Stand for the gas tank. Water bottle. Receiving and distributing tubes. Stopcock. Needle for injection.

does not recommend this plan of procedure in acutely inflamed uterus, vagina or tubes. He claims it is not dangerous, but to most surgeons the possibility of spreading a quiet infection through the tubes and producing an active peritonitis would seem to be an ever present menace.

The pioneer work in this country for the testing of tubal patency by transuterine insufflation was done by I. C. Rubin of New York.

Undoubtedly, the use of pneumoperitoneum for radiographic study of pathological conditions within the abdomen and pelvis, which are diffi-

cult of accurate diagnosis by the usual methods of physical examination, will find its place in surgery. Whether it will prove as satisfactory as the first enthusiasm would indicate remains to be determined. We do not believe it will in any way lessen the value of the methods of physical examination usually employed for diagnosis, yet in competent hands and in properly selected cases where diagnosis is otherwise difficult or impossible it should become a very important aid.

Its employment can be made quite safe by limiting the amount of gas injected, by making the injection at points where no large vessels lie, and away from cicatrices where the intestines may lie in contact with the abdominal wall. The attachments of a tumor to the abdominal wall are also to be avoided.

John L. Tierney and H. H. Turner¹⁶ have shown in animals that air may be injected intravenously and roentgenograms made. These dogs suffered no material inconvenience, and they state that they have injected oxygen into a vessel on one side and carbon dioxid on the other side of the animal's body in order to regulate the respiratory rhythm. The dogs showed no evidence of air embolism. This experiment would appear to show that the old dread of air embolism in man is a myth. Nevertheless, it will require some temerity to make the experiment in man, until further investigation is made.

In a personal communication under date of May 10, 1924, Unger,¹⁸ of New York, from his experience in transfusion, confirms the observation of Turner. He says that "small amounts of air can be injected intravenously without any danger of air embolism. Coughing is the only discomfort that I have seen following even several small doses. The old theory that the injection of air intravenously would cause death is erroneous. I wish to make it perfectly clear, however, that I do not advocate the intravenous injection of air. I wish to state most emphatically that blood transfusions can and should be performed without air being introduced. If any air is injected it is due to careless and faulty technic."

If these experiments are correct, there will be little danger from puncture of a large venous sinus when injecting the air. Still it might be well to make the puncture at some other less vascular spot.

The procedure is indicated when the diagnosis of intra-abdominal or pelvic lesions is not clear after physical examination. The outline of the liver, spleen, kidneys, uterus, bladder, tumors of various kinds, inflammatory disease of the uterus and adnexæ may in certain cases be visualized by the x-ray and considerably clarified.

Pregnancy, it is claimed, can be determined much earlier by this

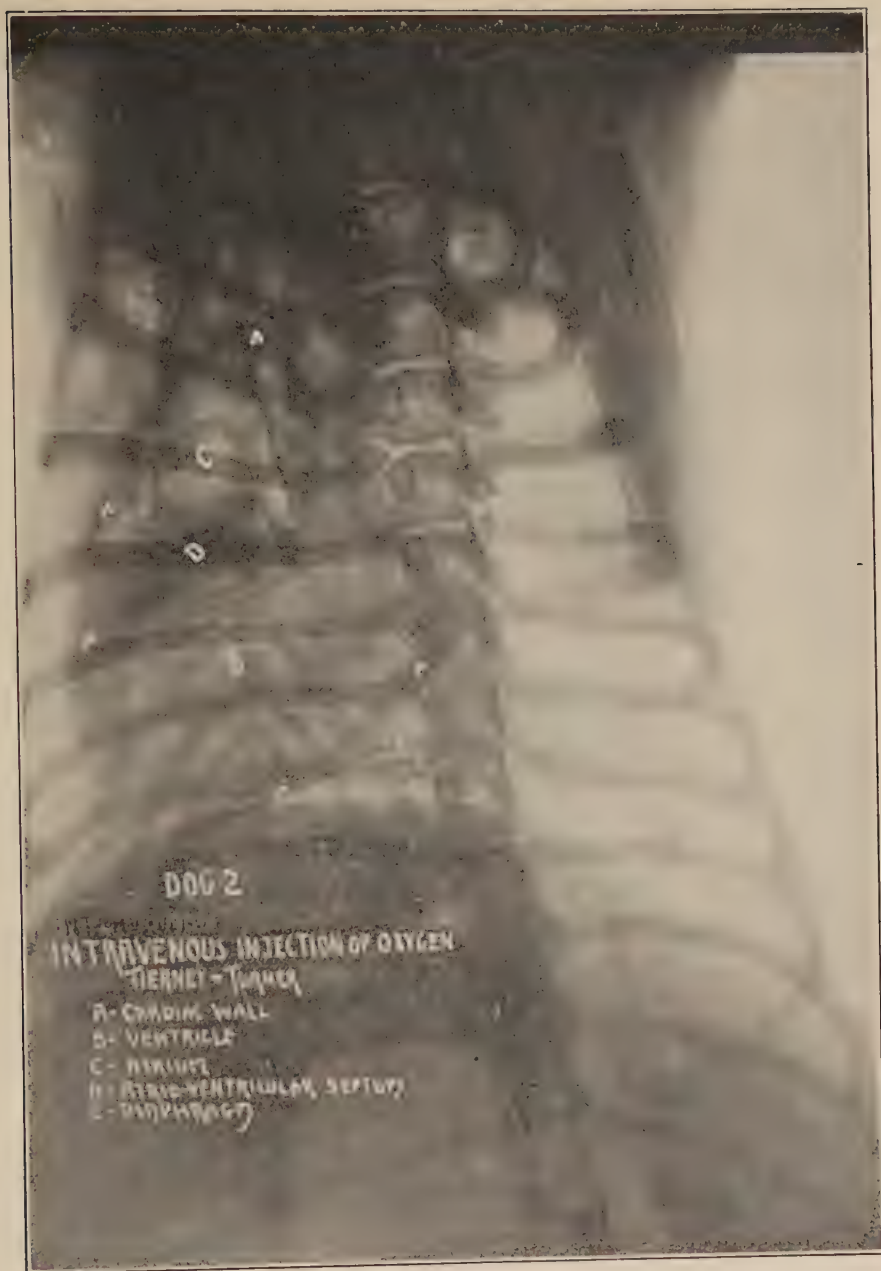


FIG. 27.—ROENTGENOGRAM OF DOG AFTER INTRAVENOUS INJECTION OF AIR.
(Tierney and Turner.)
The dog was alive when the plate was taken.

method than by simple skiagraphy. Peterson claims to be able to differentiate pregnancy from fibroids in the early months of gestation by the broadening of the lower segment of the uterine body. It is probably true of the claims made that the ossification centers in the bones of the fetus can be detected much earlier by this method. Some have claimed to be able to tell the dead fetus by the overlapping of the bones of the skull, which takes place when death of the fetus occurs. Turner has not found this to be so accurate in his work. The determination of deformed pelvis and particularly of disproportion between child and pelvis can be determined.

The employment of air injections into the intestine carefully given, in conjunction with pneumoperitoneum, is said to enable the roentgenologist to show the outline of the wall and lumen of the intestine. This may prove of considerable value. It should not be overlooked, however, that when injected into the bowel under too great pressure, there is danger of rupture.

Discomfort and Dangers.—When the injection is made into the the peritoneal sac, pain is elicited as soon as distention occurs as shown by the manometer reading. If the pressure is kept up the pain may continue for some time. It is less from carbonic acid than from oxygen because of the prompt absorption of the former. The use of a sedative prior to injection quiets the mental excitement and fear and perhaps benumbs the actual perception of pain.

In cases in which the air has been injected into the subperitoneal space a marked emphysema may result. This occurrence can readily be discovered if slight pressure of a finger is repeatedly made on each side of the needle. Under these circumstances crepitus from the local emphysema is detected at once and the injection is discontinued until the operator is certain the needle has entered the cavity.

Another method of determining that the needle is not in the peritoneal sac is by the sudden increase in the manometer reading when the gas is delivered extraperitoneally.

Frik⁴ observed a case in which the pneumoperitoneum was followed by emphysema of the neck involving the deeper layers between the muscles. He explains this occurrence by assuming that the air is forced upward toward the neck through the retroperitoneal tissues and mediastinum. He seems to think such occurrence cannot be avoided with certainty.

Care in the technic, slow inflation, and observation of the manometer and careful guard against emphysema at the point of puncture will generally prevent this accident.

Distention causes peritoneal shock if the tension is increased too

rapidly, or if carried to the point where the pressure is too high. When the discomfort is noticed some air or gas may be allowed to escape to lessen the tension.

In the light of the observations above, air may be passed into a vein up to the point of the oxygen absorbing capacity of the blood. Beyond that point it may impede the circulation seriously.

Fluoroscopic readings are very valuable. Both these and the roentgenograms require experience to obtain correct and accurate interpretation.



FIG. 28.—ILLUSTRATING DR. TURNER'S CASE IN WHICH AN ABSCESS IN THE ABDOMINAL WALL DUE TO PNEUMOCOCCIC INFECTION WHICH HAD NOT BEEN POSITIVELY DIFFERENTIATED CLINICALLY WAS CORRECTLY INTERPRETED.

Note the thickened abdominal wall. Outline of abscess. Intestinal lumen below.

Indications.—We are able to visualize abscesses (Fig. 28) of the abdominal wall, adhesions between the viscera and the abdominal wall, the diaphragm, subdiaphragmatic areas, the liver, perihepatitis, adhesions to the diaphragm, the kidneys, the spleen, omental fixation, bands of adhesion, omental and mesenteric tumors. We can also delineate the hydropic gall-bladder, the duodenum, the intestine, the stomach (after filling with air), gastromural infiltration, the thickness of its walls, and the colon. By pelvic examination can be observed the air-distended bladder, intravesical papilloma, the female internal genitalia, uterine and ovarian tumors, and the amount of their pedunculation. Inflammatory

masses in adnexal disease and intraligamentous masses are well shown. It is useful in showing ectopic gestation sacs, those which reach the period of ossification of the fetal bones and particularly those rare but sometimes obscure cases which have gone beyond the normal term of gestation.



FIG. 29.—NORMAL SIZED UTERUS, TUBES AND OVARIES IN YOUNG GIRL. (After Turner.)
Slight displacement to the left. Louisville City Hospital, Case No. 13246.

Peterson¹¹ claims that the diagnosis of pregnancy can be made very early by showing the broadening of the lower segment of the uterine body.

The use of barium or bismuth in the gastro-intestinal tract as is usual for roentgenologic examinations may be made a complement of this method.

Preparation of the Patient.—The preliminary preparation is simple. When an examination is to be confined to the pelvis it is only necessary to give a couple of cleansing enemas. For a general abdominal examination a laxative is given the night before, and a cleansing enema two hours prior to the examination. In more nervous individuals who have

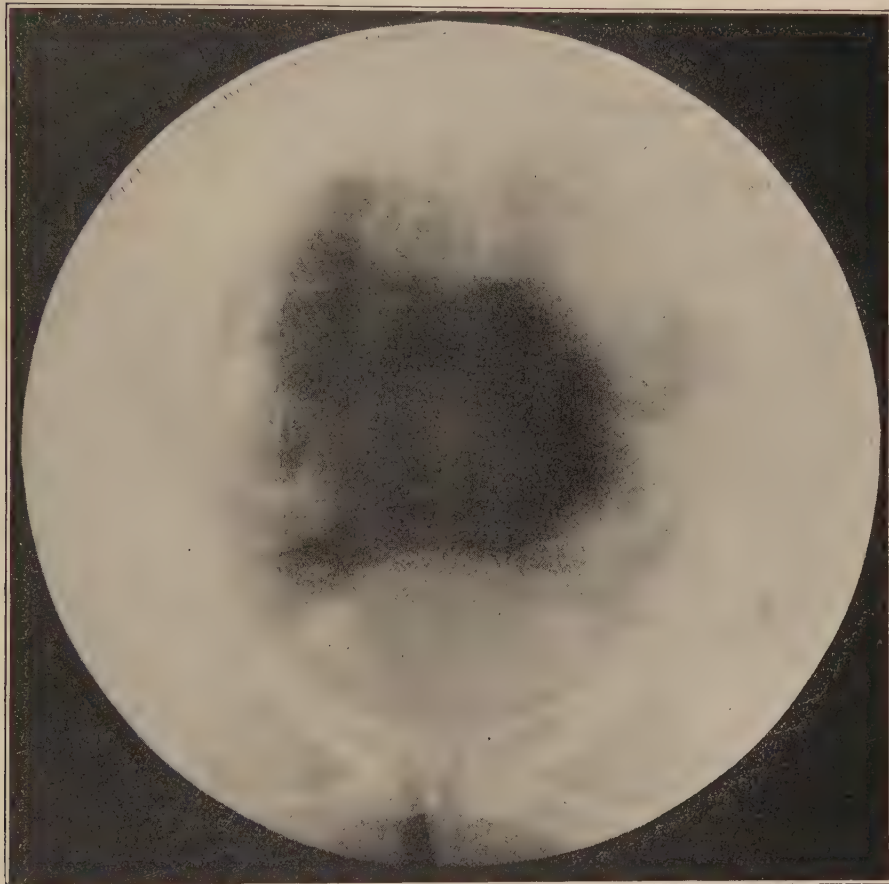


FIG. 30.—PNEUMOPERITONEUM. CONGESTED UTERUS. RIGHT PYOSALPINX. LEFT PYOSALPINX. (After Turner.)

no idiosyncrasy to its use a sedative may be given half an hour before the observation is made.

Position of Patient.—The patient, with the abdomen insufflated and the needle withdrawn, is placed in the prone position. For pelvic examination the pelvis is elevated. For lateral observation the patient is placed in the supine position with the tube at the side. For upper abdominal examination the position should be a reversed Trendelenburg.

To examine the stomach a Seidlitz powder in two parts is administered after the patient is on the table. The intestine may be outlined with air before examination. The same is true of the bladder.

Vaughan and Rudnick¹⁹ recommended the employment of air in the bladder in conjunction with skiagraphy to determine the presence of traumatic rupture of the organ. In intraperitoneal rupture the air may be observed in the peritoneal cavity. Where the wound is extraperitoneal the air is shown in the cellular spaces of the pelvis. They conclude that it is a positive diagnostic measure and free from risk.

DIRECT INTRAPERITONEAL LAPAROSCOPY.—Jacobaeus⁶ first considered the possibility of utilizing cystoscopic methods of examination of serous cavities in 1910. He has demonstrated diseases of the pleura,

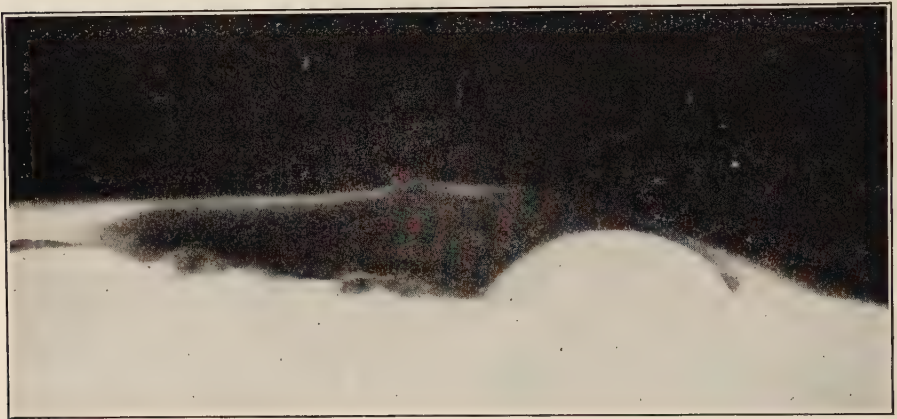


FIG. 31.—SHOWING 3-MONTH PREGNANT UTERUS, LATERAL VIEW.

Note umbilicus, anterior abdominal wall, differentiation of intestine. (Louisville City Hospital, Case No. 10906L-5-15-1923.)

after air inflation, by means of a cystoscope, using a cannula and passing the cystoscope through its lumen.

The same method may be employed in the abdominal cavity. The laparoscope is fitted with an automatic valve which prevents the escape of air. The instrument is inserted after the skin is cocainized and incised for a short distance so the trocar can enter without producing pain. By direct laparoscopy the peritoneopathies, such as tuberculosis of the peritoneum, bands, the gall-bladder, the under surface of the liver, the uterus, tubes, and ovaries may be seen. A very beautiful picture is presented. This may prove of much value in a very limited field. The proceeding must be conducted under scrupulous asepsis.

Orndoff has also made use of this method.

X-RAY METHOD FOR OUTLINING GALL-BLADDER

In spite of considerable improvement in technical procedures and apparatus, the x-ray diagnosis of gall-bladder conditions has remained decidedly unsatisfactory, both to the roentgenologist and to the surgeon. It is probable that with the best technic at the present time it is possible to demonstrate in the neighborhood of 40 per cent of gall-stones on the x-ray film.

George and Leonard first called attention to the fact that it was very often possible to show the outline of the gall-bladder upon the film even in the absence of any shadows suggestive of stones. Upon the basis of a rather large series of cases checked by operation, they concluded that the x-ray demonstration of gall-bladder outline was in itself sufficient to indicate pathology in that organ. They feel that it is even possible to demonstrate an abnormal gall-bladder when the stones which it contains cannot be visualized. The report, therefore, upon such an x-ray finding is, "probable gall-bladder pathology with, or without, stones."

Much and often valuable information as to the status of the gall-bladder is also obtained from x-ray examination of the stomach and duodenum by means of the opaque meal. Roentgenologists with a large gastro-intestinal experience feel that there is a certain rather characteristic group of x-ray signs which, taken together, form valuable indirect evidence of gall-bladder disease. These include pylorospasm, fixation of the pylorus and duodenum high in the right side, indentation of the lesser curvature surface of the duodenal bulb, certain types of bulb deformity due to adhesions, and interference with normal motility, usually consisting in late emptying of the stomach. It is thought that these signs, especially in conjunction with the visualized gall-bladder, offer very valuable and reliable, positive evidence of gall-bladder disease. However, when absent, the negative evidence is of little value, as it is conceivable, and has been proved by large series of operative cases (Mayo Clinic, Carman, *Radiology*, 1924) that extensive gall-bladder pathology may exist without the presence of either direct or indirect x-ray signs.

In February 1924, Graham and Cole reported (*Journal of the American Medical Association*) a series of gall-bladder cases examined by a new method. This procedure is based on the fact that some dyes are excreted entirely through the liver, in the bile. Following a lengthy period of animal experimentation, the dye finally chosen was tetrabrom-phenolphthalein. In the technic originally described this dye was used in the



FIG. 32.—GALL-BLADDER OUTLINED BY TETRABROM-PHENOLPHTHALEIN.
Twenty-four-hour observation. (Case of Dr. C. D. Enfield; method of Graham and Cole.)

form of a calcium salt, 5.5 grams of the dye being combined in about 400 c.c. of solution with calcium lactate and calcium hydroxid.

The examination was undertaken early in the morning, the patient having omitted breakfast. At this time two or three films were made over the gall-bladder region to show whether the unprepared gall-bladder could be visualized and whether there were any demonstrable stone shadows. The solution of tetrabrom-phenolphthalein was then slowly injected intravenously, stopping the injection for a few moments if vertigo or nausea occurred. The patient was encouraged to be on his feet as much as possible during the next few hours. For luncheon a glass of milk only was permitted. The evening meal consisted solely of carbohydrates. Films were made routinely at four hours, eight hours, thirty-two hours, and forty-eight hours.

In the first series of cases reported the authors felt they had established a normal performance for the gall-bladder under this treatment, and that from such a series they were justified in drawing certain conclusions as to the functioning ability of the organ. Normally the gall-bladder shadow was very distinctly visible in the four hour film and continued so in the eight and twenty-four hour films. At either thirty-two or forty-eight hours it was no longer visualized. The normal gall-bladder in these films should show a variation in size and density and a consistently regular contour. The maximum density occurred at either eight or twenty-four hours. The maximum size apparently might occur at any period when the shadow was shown.

Such a normal sequence proves that the cystic duct is patent; otherwise the dye would not have been able to enter the gall-bladder at all. It further proves that the organ is capable of emptying itself, that the walls are not greatly thickened or infiltrated, nor greatly bound down by adhesions, else normal motility would be interfered with. Emptying at thirty-two or forty-eight hours probably indicates that there is no marked stasis.

Thus far this procedure apparently establishes a normal and, by inference, an abnormal standard. Closer interpretation of the abnormal findings will have to wait on more experience with the method. It would appear at present that complete failure to visualize the gall-bladder following injection is presumptive evidence of either gross impairment of liver function or occlusion of the cystic duct. It would seem that persistence of the shadow long beyond the thirty-two hour period would indicate biliary stasis. Where adhesions are suspected a persistent irregularity in the outline of the shadow would certainly offer valuable confirmatory evidence. The method is of further value in

enabling the roentgenologist to visualize certain gall-stones which he is unable to show without this preparation, since they appear in the prepared gall-bladder as negative shadows of decreased density.

More recently an improvement in the technic has been made by the use of a concentrated solution of the sodium salt of the same dye. This substance, ready prepared, is obtained as the sodium salt of tetrabrom-phenolphthalein. Five and one half grams are dissolved in 40 c.c. of distilled water and the mixture sterilized for fifteen minutes over a boiling water bath, for the patient of 125 pounds or over. If the patient weighs less than 115 pounds a corresponding reduction in the amount of dye used is made. The 40 c.c. of solution is divided into two equal parts. Twenty c.c. are injected intravenously early in the morning, the patient, as before, omitting breakfast. One half hour later the remaining 20 c.c. are injected. Considerable care must be used in the injection as necrosis follows any infiltration of the tissues outside the vein. As in the original technic, preliminary films are made before injection and films are again made at four, eight, twenty-four, and thirty-two hours.

Routine instructions for the patient are as follows:

1. Omit breakfast.
2. Appear at laboratory for preliminary films and injection.
3. Omit lunch, except that a glass of milk may be taken if hunger is very pressing.
4. Omit proteins from evening meal.
5. Lie on the right side at all times when lying down. It is preferable to be up and about a good deal of the time.
6. Take 40 grains of sodium bicarbonate in water every three hours for forty-eight hours following the injection (patient not to be wakened for this).
7. There is no restriction as to the amount of water that may be consumed.

The shadows with the sodium salt are possibly a little less dense than with the calcium salt. The decrease in the amount of fluid to be injected and the simplicity in preparation are, however, more than sufficient to offset this slight disadvantage. There is, after all, no point in obtaining a very dense shadow as long as the gall-bladder outline is clearly visualized.

The only disadvantage to the method lies in the fact that there is a rather constant nausea during a good portion of the day of the injection. There may also be some vertigo. The nausea is not ordinarily severe, and the discomforts to the patient of the method may perhaps be

fairly compared with those attending the so-called drainage of the gall-bladder with the duodenal tube by the Lyon's method.

The accumulation of material and the deductions to be drawn from later reports of the various workers now using this method will eventually establish its definite value.

This method of gall-bladder examination is still in the developmental stage, neither the drug nor the method of administration having been finally determined upon.

A more recent development is the use of 3 Gms. of tetraiodophenolphthalein in 12 per cent aqueous solution intravenously. Early reports indicate that this dye in the smaller amount gives equally good shadows with the bromin compound and with somewhat less general reaction and less danger of necrosis of the tissues, should infiltration occur.

Also, a number of investigators have used both the bromin and the iodine compound through the duodenal tube and have administered both dyes orally in formalinized or salol coated capsules. For oral administration the dose of the bromin compound should be about 7.5 Gms. divided into a number of small, hardened, or salol coated capsules and given during the evening meal. The iodine dye may be given in the same way, the dose being about half that of the tetrabrom-phenolphthalein.

Results from oral administration are naturally less uniform than from intravenous, the only advantage being lessened reaction. It seems probable at the time of writing that intravenous administration of the tetraiodophenolphthalein will finally be the method of choice.

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CHAPTER IV

INFLAMMATION OF THE PERITONEUM

CLASSIFICATION

Classification.—Inflammation of the peritoneum may, for purposes of clarity, be divided:

1. According to extent, into circumscribed or local, spreading or general.
2. According to method of production into traumatic, non-traumatic.
3. According to bacterial flora into staphylococcic, pneumococcic, streptococcic, gonococcic, tuberculous, that due to *Bacillus coli*, etc.
4. According to severity, into acute, subacute, and chronic.
5. According to the reaction of the tissues into dry or plastic, serous, serofibrinous, seropurulent, purulent.

The term fulminant (septic) is applied to a rapidly progressive type. In some of these cases the patient dies within forty-eight hours of the onset, even before suppuration begins.

The term hemorrhagic is applied to those acute forms with marked extravasation of blood into the membrane and the underlying tissues.

Perforative peritonitis is applied to the type resulting from the wounding or rupture of any of the hollow viscera with the escape of noxious material into the cavity, also to those violent cases where an intensely virulent organism has infiltrated the walls of one of these organs, and by contact has infected the peritoneum without a manifest or demonstrable gross lesion.

Septic Peritonitis and Suppurative Peritonitis.—Much confusion has arisen in the use of these terms. They are identical etiologically, but differ in that septic peritonitis is generally diffuse and leads to a rapidly fatal termination, often before pus formation has time to occur.

ETIOLOGY

The exciting factor in the causation of this affection is an invasion of the peritoneal sac with living microorganisms. We may admit for the sake of argument that certain inorganic and organic chemical substances excite a marked local reaction, which was thought by many to be a true inflammatory process. The fact that this process is always localized to the immediate site of the traumatism and progresses promptly to repair

except in those cases in which a large amount of tissue destruction occurs, permitting bacterial invasion from the adjacent hollow viscera, only emphasizes the contention that clinically peritonitis is always bacterial in origin.

The varieties of bacteria most frequently acting as causative agents are quite numerous, the most important being:

1. Streptococcus
2. Bacillus coli communis
3. Staphylococcus
4. Bacillus pyocyaneus
5. Pneumococcus
6. Gonococcus
7. Bacillus tuberculosis.

In 100 autopsies collected from the Louisville City Hospital records by Dr. R. P. Ball of the Medical School, Pathological Department, University of Louisville, in cases in which peritonitis figured prominently, 49 were males and 51 females. Of these cases 33 showed localized peritonitis and 67 were general in type.

With reference to the causative organism in this series, the bacterial flora of the mixed infections were as follows:

Mixed Strains

	<i>Cases</i>
Streptococcus mixed with other organisms.....	22
Staphylococcus mixed with other organisms than streptococcus....	7
Bacillus coli mixed with other organisms than streptococcus.....	3
(Bacillus coli mixed with other organisms including streptococcus, 17)	

Pure Strains

Streptococcus	18
Bacillus coli	7
Staphylococcus	4
Tubercle bacillus.....	5
Bacillus typhosus.....	3
Gonococcus positive bacillus.....	1
Gonococcus	1
Pus not examined.....	29
Total	100

A mixed infection is not infrequently seen in acute cases, and is, as a rule, particularly virulent.

The causes which predispose to the development of peritonitis are trauma, external and internal, rupture of the gut from gunshot or stab wounds, or from compression violence damaging the hollow viscera, rupture of the bladder from distention or trauma, overdistention of the gall-bladder with rupture, ulceration through its walls as a result of stones, gangrenous inflammation with infection of the peritoneum from contact or from leakage. Healthy bile appears to be harmless to the peritoneal structures, but since lesions of the gall-bladder nearly always show the presence of infection, the bile becomes a source of contamination.

One of the most frequent sources of contamination is the inflamed appendix, which being a rudimentary organ resists infection poorly, and bacteria infiltrate its walls to infect the adjacent peritoneum or the invasion takes place through rupture or gangrene of this organ, or of an abscess surrounding it. Next in frequency the infective agent enters the cavity through the fallopian tube, or through its actively inflamed wall. Infection in puerperal cases or following abortion may also enter through the uterine wall or by contact with cellular tissues of the parametrium. Rupture of a gastric or duodenal ulcer quite frequently is the means of contamination. Infection may also occur in cases of ectopic gestation in which invasion of the extravasated blood with bacteria occurs either from the intestine or from the uterine adnexa, also from rupture of the uterus in labor or from instrumentation. Typhoid fever is an occasional cause of this affection. Rupture of one of the ulcers permits a bacterial flux into the cavity. Internal and external hernias, when strangulated, become prolific sources of contamination. Internal ileus, volvulus, intussusception, thrombosis of the mesentery, are all active predisposing causes. Ulcerations of the intestine, either from tuberculosis or malignancy, may result in peritonitis. The extension of suppurative infection in adjacent organs, abscess of the liver, perineal suppuration, phlegmonous cellulitis, Pott's disease, etc., may cause it. Influenza in the more severe forms frequently affects all of the serous membranes, and we have personally observed suppurative pleurisy, pericarditis, and peritonitis in the same individual suffering from influenza.

In some of these cases there seems to be no demonstrable point of invasion, and it is probable that the contamination is from a blood stream infection.

That the causative agent in peritonitis is always the presence of bacteria is generally accepted. Formerly the pathologists recognized the chemical form of peritonitis because in certain cases no bacterial flora was obtained from the fluid. It is now believed that although bacteria

may not be found in the fluid in certain cases, they may be demonstrated within the fibrinous exudate covering the coils.

The exact relative frequency of occurrence of different causative organisms cannot be given because of the great variation of the type of bacteria in different series of cases and of the difference in the methods of taking cultures. Deadhouse studies are not so accurate as those made upon the living subject, since there is likelihood of postmortem contamination. The colon bacillus is claimed to contaminate the inflamed peritoneum within two hours after death.⁵

Certain cases of peritonitis have shown pure strains of bacteria, which have been taken to be the causative factor. The streptococcic group shows the largest number in pure culture, particularly in cases observed at necropsy. The staphylococci play a more important rôle than has been believed. The larger number of cases show mixed strains of bacteria.

Notwithstanding numerous efforts to demonstrate a specific organism as the cause of peritonitis, particularly that resulting from appendicitis, this has been accomplished only for individual cases. The present opinion is that any one of the pyogenic organisms may be the cause in the production of peritonitis. The organisms may appear in pure strain, but more frequently a number of different organisms are present. The mixed cultures are believed to produce a more virulent type of infection. The colon bacillus is the most frequent contaminating organism.

Weil¹⁰ found that in cases of peritonitis following appendicitis this organism was present in 60 per cent. In 19 per cent of the cases it was accompanied by streptococci.

It is a well-known bacteriological fact that certain organisms outgrow others in mixed cultures. This, however, does not *a priori* establish the more active organism as the original cause of the disease. Rost⁹ thinks that only the bacterium toward which the body develops antibodies, as agglutinins, should be considered as the cause of the disease.

It is doubtful if the determination of this is positive except in cases of blood stream infection. Putrefactive bacteria are often added to the other strains in peritonitis. In fact they are often present in lesions near the alimentary canal. They tend to produce the offensive odor found in abscesses in this region. Some observers claim that their presence indicates an increased virulence, particularly in cases following appendicitis.

Heile⁸ found in a large number of his cases an anaërobic spore bearing bacillus with marked necrotizing and toxic properties.

Bacteria of a similar type may account for the tendency to rapid development of gangrene in certain inflamed appendices. The writer has

believed that the gangrenous process results from impairment of the circulation in the walls of the appendix when the lymph vessels become choked with bacteria. Such an occurrence may be found as the result of any bacterial infection. Undoubtedly the mixed types are more likely to terminate in necrosis. The pneumococcus and *Bacillus Koch* are less likely to cause necrosis of the appendix.

The virulence of peritonitis varies greatly with the location of the causative lesion. Some of the most severe types occur from puerperal infection. Next in severity are those in which the bacteria gain entrance through the appendix.

Peritonitis from perforations of the duodenum, according to Brunner,¹ are more dangerous in cases in which there is absence of hydrochloric acid in the gastric secretion as in carcinoma.

Gruber² and Durham, Buxton, Dudgeon and Sargent have shown that a very short time after virulent bacteria are injected into the peritoneum the fluid may be free of the organisms, while upon the surface of the omentum and intestine the bacteria are found in a thin coating of fibrin.

Murphy⁸ states that, "the introduction or penetration of bacteria into the peritoneal cavity is not sufficient to produce peritonitis if that membrane be healthy and free from foreign bodies." This is in accordance with the findings of other writers.

When the peritoneal basal membrane is damaged, however, peritonitis is likely to result. There must be a point, however, as shown clinically at which the dosage and virulence of the invading organism overcomes the resistance of this structure and peritonitis is set up. That some bacteria gain entrance into the peritoneum in almost every laparotomy must be admitted. When the number and virulence remain small, peritonitis does not result. Occasionally, however, a large dose in a patient of feeble resistance causes peritonitis.

Basing our consideration upon the premise that all cases of peritonitis are bacterial in origin, the cryptogenic type of infection, in which the route of invasion of the bacteria cannot be determined, remains for explanation. Such cases are viewed by pathologists as doubtful and they look upon them as errors from failure to observe very minute perforation either upon the operating or necropsy table.

Cases are observed, however, in which the most careful investigation fails to reveal even the most minute leak. Undoubtedly a small number of cases of peritonitis are the result of blood stream infection. Clinical evidence is strongly in favor of this conclusion, but the actual production of peritonitis by this route is somewhat difficult of demonstration.

The typical cases of peritonitis are those resulting from a leak, as

from ruptured duodenal, typhoid or other ulcer, a gangrenous gall-bladder or appendix, or an inflammation of these or similar organs in which the bacteria grow through the wall and by contiguity invade the peritoneum. Similar invasion occurs through the softened macerated uterine or tubal wall in cases of violent infections of these structures.

Occasionally acute gonococcus peritonitis occurs from invasion by the gonococcus through a tube which shows but little inflammatory reaction, while the uterine mucosa may appear healthy. The same is true of pneumococcal infection.

The possibility of bacteria invading the peritoneum through healthy hollow viscera is unsettled.

Jensen,⁴ by feeding two guinea pigs with virulent pneumococci, gave rise to a fibrinous peritonitis. The intestinal wall was loaded with organisms although no ulcerations were present in the mucosa. Because pneumococcic peritonitis so frequently begins with diarrhea, Jensen believes that the invasion takes place from the alimentary canal.

J. E. McCartney⁶ has made a rather comprehensive study of the pathogenesis of pneumococcic peritonitis. He concludes that it is difficult to correlate the hematogenic theory with the clinical facts. Adults are immune and the disease does not occur in young babies who frequently suffer from respiratory pneumococcal infections. If blood borne, he asks why the disease affects only girls at certain ages. He claims that primary pneumococcal peritonitis begins in the pelvis, the most resistant portion of the peritoneum, and argues from this a tubal as against a hematogenous origin of the infection. "Pneumococci can be isolated from the blood in practically every case of pneumonia, and yet pneumococcal peritonitis is an exceedingly rare complication.

"Rolleston (1908) found only 11 cases in 4454 cases of pneumonia, or 0.25 per cent, and Rischbieth (1910) records that in 6000 cases of pneumonia peritonitis was a complication in only one instance."

McCartney states that the intestinal theory is untenable from clinical, bacterial and experimental evidence, and concludes that the infection reaches the peritoneum from the vagina by way of the genital tract.

"The clinical evidence is based on the following observations: (a) The disease occurs only in young girls; (b) the majority of cases occur between the ages of 3 and 7 years; (c) unlike pneumonia, infection is more common in the summer months; (d) the girls belong to the lower classes; (e) such children frequently suffer from vulvovaginitis, and the vaginal secretion may be neutral or even alkaline in reaction; (f) virulent pneumococci may be isolated from the vaginal secretion of these children; (g) the disease occurs suddenly in healthy children, the first

symptom being subumbilical pain; and finally (*h*) on laparotomy, it can be shown that the disease begins in the pelvis, and in early cases is confined to the lower portion of the abdomen.

"The bacteriological evidence is based on the following findings: (*a*) Pneumococci were isolated from the vagina in all cases of the disease examined, the organisms always being of the same type as the one causing the disease: (*b*) the pneumococci in the throat may be a different type from those found in the peritoneal cavity, thus showing that the path of entrance of the infection was neither the pharynx nor the intestinal canal; (*c*) at autopsy, the peritonitis was the only lesion present.

"Furthermore, the animal experiments demonstrate that: (*a*) The disease can be reproduced in young monkeys by vaginal inoculation with pneumococci; (*b*) an alkaline reaction favors infection; (*c*) the organisms ascend the genital tract and do not cause symptoms until they reach the peritoneal cavity."

While the weight of opinion appears to favor the contention of McCartney, the author recognizes the possibility of blood stream infection in certain cases of pneumococcic peritonitis.

Zagari⁷ fed healthy dogs with tubercle bacilli and found they could penetrate into the circulation without an evident lesion of the intestinal wall. Such observations are always open to question. The fact that cells enclose and carry tubercle bacilli and that such cells may enter the vessels without a break in structure makes such a mode of invasion possible.

The reaction of the peritoneum to the presence of bacteria resembles that which occurs from the presence of any foreign body. In the case of bacterial invasion, however, the process is progressive and tends to spread widely and is not localized as is the result of a simple irritant. The character of the reaction varies with the type, dosage, and virulence of the organism, also with the character and the location of the causative lesion.

The phagocytic action of the cells is also important in protecting the tissues from invading organisms. It has been shown that after foreign material enters the peritoneal cavity an initial leukopenia in the peritoneal fluid occurs which lasts from one to three hours.⁷

There is some difference of opinion concerning the interpretation of this occurrence. It seems likely that it results from the temporary overwhelming action of the bacterial toxin upon the cells. Murphy states: "This explosive destruction was real, for a microscopical examination of the omentum showed no organisms on its surface."

Particles of bacteria become encircled by the cells of the peritoneal

fluid, macrophages, within a short time after the original contamination. These cells are present even before the development of leukocytosis, which occurs in from one to three hours. They are so choked with bacteria that the cytoplasm may scarcely be seen.⁷ At the end of three hours the polymorphonuclear leukocytes appear in large numbers and actively incorporate the foreign particles. In twenty-four hours large cells (macrophages) appear in great numbers and incorporate both the leukocytes and the bacteria. A similar process takes place in the later stages when purulent accumulations are to be removed.

PATHOLOGY

The pathological changes in acute peritonitis depend upon its causative condition and the amount of damage done at its inception. Following upon traumatism, rupture of the intestine, urinary bladder, gall-bladder, stomach, an acutely inflamed appendix, or of an appendicular or other abscess which carries a bacterial flora into the abdomen, there promptly ensues a marked reaction. The amount of this reaction depends upon the dosage, variety and virulence of the bacteria, the coincident damage to the peritoneal structures and the resistance of the individual to such contamination.

Notwithstanding the usual belief that the peritoneum is peculiarly susceptible to infections, it is a fact that the unbroken peritoneum will withstand certain organisms particularly well. It has been established⁴ that the injection of pure cultures of staphylococci into the healthy peritoneum of rabbits or guinea pigs produces but little reaction. A culture of tubercle bacilli on the other hand is promptly followed by the development of tuberculosis. When pure cultures of pus-producing organisms are placed in contact with the broken peritoneal surface an inflammatory process is immediately excited.

It is claimed by some observers that the reason for the failure to develop peritonitis after the injection of pure cultures of pus-producing organisms in animals lies in their rapid absorption from the peritoneal cavity. Somewhat in confirmation of this contention it was found at Cornell University Medical School that following the injection of pure cultures of virulent pus-producing organisms into the peritoneum of a healthy animal these organisms could be detected in the blood-vessels within five minutes.

Johnson suggests that this occurrence may change the accepted views that the symptoms grouped under the term *sapremia* and considered due to the absorption of toxins, in reality are cases of blood stream infection.

Under present laboratory methods this contention may be promptly decided by taking a culture of the blood.

It is a generally recognized fact that a mixed bacterial flora is more likely to excite a peritoneal inflammation than a pure strain.

Promptly after contamination of a damaged peritoneum, particularly if it occurs under pressure, there is an increased supply of blood to the part. Almost immediately the peritoneal surface becomes dry and glazed. This is soon followed by an exudation of plastic lymph in the effort to protect the structure against further insult, also to envelop and destroy the invading organism. A large number of leukocytes are hurried to the locality to attack the invaders. In the case of success in this process the reaction subsides and the part is soon restored to normal. This occurs when the virulence and dosage of bacteria are small. When the virulence is great and the leukocytes are overcome the process continues with increase in the swelling, a multiplication of fixed tissue cells with the transudation of serum. This free serum varies very greatly in different cases, the amount being small in certain mild types of infection and quite large in more severe types. This fluid soon becomes flocculent from the presence of dead cells, fibrinous material, and bacteria, and in many instances assumes a seropurulent form. Certain bacteria show a marked tendency under certain conditions to excite the production of a large amount of serous exudate. This is particularly true of tuberculosis as shown in the disseminated granular type. On the other hand in some cases of the same infection only a small amount of serum forms, as in the serofibrinous type. Again, the tuberculous process may produce only a fibrinous exudate, as seen in the plastic type.

Gonococcal infection, usually more acute, also appears primarily to produce in some instances a very free outpouring of serum from an intensely reddened peritoneal surface. The writer has observed a number of cases of this type at operation performed immediately following the onset of the disease in which the causative bacteria were recovered in pure strain from the serum.

As long as the strain remains pure, there appears to be less tendency for the serum to become purulent than in other forms of infection. The gonococcus seems to be a slow pus-producer in the peritoneal structures except when mixed with other bacterial flora. It has a greater tendency to excite the production of plastic exudate.

The pneumococcus also has a tendency to excite the formation of plastic material and localized abscesses. Most cases of severe peritoneal infection from this organism with coincident infection of the pleura and pericardium and the presence of a large amount of free fluid in each sac

show mixed strains. The pneumococcus in these cases is accompanied by the streptococcus, the influenza bacillus or other organism. A blood stream infection is demonstrable under these conditions, and no positive route of peritoneal invasion may be demonstrated.

Other bacteria, as staphylococci and colon bacilli, appear to excite a more localized reaction, greater necrosis of tissue and tend to form localized collections of pus. Such an abscess results from death of leukocytes and fixed tissue cells as the result of the toxic bacterial poison and from interference with the local circulation. The bacterial products have a peptonizing power which softens the exudation and a yellow fluid forms which carries the dead cells and certain detritus.

A temporary amelioration both of the pathological process and the clinical symptoms follows the enclosure of an appendicular, duodenal or cholecystic leak by a protective wall. Failure upon the part of the protective forces permits rupture of the localized abscess and a further extension of the process into the acute spreading form of inflammation. This follows rupture of the abscess sac from overdilatation or trauma. When this occurs, and also in certain cases of virulent contamination, the inflammatory process shows a remarkable tendency to extend widely over the peritoneal surface. There is still present a contest between the invading organisms and the host in which new lines of defense are formed. This is evidenced by the agglutination of intestinal coils and the formation and isolation of local collections of pus. This defense process may yet protect a portion of the peritoneal surface from involvement. In other instances the defensive mechanism fails to form and the patient succumbs to the intense generalized sepsis.

Pockets of pus may form in any portion of the peritoneal cavity quite distant at times from the original point of leakage. These are seen in the pelvis, loins, and subdiaphragmatic regions. Naturally one expects to find the pus collections in the neighborhood of the original lesion and this is the usual location. Most of these abscesses empty into the intestine when not emptied surgically, but it is undoubtedly true that the peritoneum may dispose of small accumulations of pus by absorption.

The picture presented by the inflamed peritoneum varies greatly in different cases. The most accurate observation upon the tissue changes are those made upon the operating table, since the process is seen in its every variety and at every stage.

Necropsy findings are valuable, but show only the end results of the process when the reparative forces of the organism have been overcome by the disease.

The great importance of visceral leaks in the determination of the

development of peritonitis is shown by its frequency of development after intestinal wounds and its rare occurrence when the intestine is intact.

Perforative lesions give rise to the more acute type of the disease. One of the most frequent and typical examples of this type is that resulting from acute appendicitis. The most violent attacks of appendicitis occur in young persons. The vessels of the appendix become choked with bacteria and lymphocytes. The nutrition of its walls is impaired. The causative organisms multiply very rapidly and involve all the coats of the organ. A localized peritonitis over the appendix occurs, followed in a short time by necrosis of the entire thickness of its walls. In some instances, even before the necrosis occurs in so-called fulminant cases, the adjacent peritoneum becomes dry, glazed, reddened, while the amount of peritoneal fluid is much increased. This fluid soon becomes yellowish in color and contains considerable detritus and active colonies of bacteria. It has a tendency to spread over the entire peritoneum with great rapidity.

In cases in which the tissue resistance is poor the local reaction is limited and the constitutional reaction is very marked. A certain proportion of cases go to a fatal termination before the local reaction has advanced to a point which would indicate such an occurrence.

A similar type of case occurs from contamination during clean abdominal sections, and also in violent puerperal infections. These cases are recognized as septic peritonitis. Suppuration has not had time to develop. The cause of death in such cases has been the source of considerable discussion. Heineke⁵ made experiments upon rabbits with a view of determining this point. He demonstrated that the circulatory disturbance in cases of perforative peritonitis is due to paralysis of the vasomotor center in the medulla oblongata. This depression results from the direct action of the bacterial products. He concludes that respiratory disturbances develop later than those of the circulation, but respiratory action ceases before cardiac failure. It is generally admitted at present that death occurs in cases of this type as the result of an intense toxemia. A factor of considerable importance which has been overlooked is the toxemia resulting from the growth of bacterial flora within the intestine. It has seemed to the author that absorption of poison from this source, in addition to the intraperitoneal poison, often turns the balance unfavorably. Some authors, in addition to the toxemia, attribute to a form of shock an importance as a depressing factor. It would appear that sudden perforations by pain and reflex nervous impulse cause circulatory depression. Such shock is usually transient and only when toxemia is added does a fatality seem likely.

Some observers attribute the occurrence of death to the toxemia resulting from virulent bacterial flora. Murphy⁷ considered that death occurred in the extremely septic cases because of the "primary overwhelming dose of toxins." He also believed that the toxins released during operative manipulations accounted for so many of the early deaths after operation for appendicitis with acute peritonitis, in which views Peiser⁸ concurred as the result of his experimental investigations.

Other observers consider acidosis and dehydration as very important in the fatal result. Crile² has made some extensive studies in connection with acidosis and diminished reserve alkalinity. It was found in his work with Menten that the H-ion concentration was increased during intense fear, intense rage, extreme exhaustion, inhalation anesthesia, in surgical shock, in hemorrhage, also near the death point in dissolution from any cause. The H-ion concentration was not increased during sleep, during opium narcosis, nor in serious and even fatal diseases, as infections, exophthalmic goiter, and cardiovascular disease. He thinks that an increase in H-ion concentration takes place in all forms of extreme exhaustion, and that the clinical results of restorative measures to counteract acidosis lend support to the laboratory conception that acidosis is the fundamental condition present in all forms of exhaustion.

Crile considers a proper electrochemical activity of the cells essential to life, also that failure of this activity and neutralization of the two portions, acid and basic, of the cell results in its death. Similarly neutralization of the cells of an animal and failure of its electric reactions result in the death of the animal. He believes that anhydremia and acidosis are important factors in death. Some observers, notably Bauer, deny the importance of dehydration as a cause of death in peritonitis.

All of the factors mentioned may have considerable importance in the production of exhaustion which in most cases precedes the fatal issue.

Friedlander,³ in his study of these investigations, has very properly considered it necessary to separate the rapidly progressing sepsis from the slowly progressing actual peritonitis. He believes that because of the disproportion between pulse rate and temperature reflex influence cannot be ignored. He thinks that peritonitis is not merely a sepsis with quantitative differences, but that in the death from this disease the complicated nervous mechanism of the abdomen is an important factor. Cases of this type which last more than a few hours show marked intestinal distention. The anatomical basis for this paralysis has been attributed by Askanazy¹ to dilatation of the lymph channels around the ganglion cells in the intestinal walls. This explanation is doubted by

Walbaum, but confirmed by Strehl.¹⁰ The consensus of opinion at present is that the intestinal paresis is of toxic origin.

Hotz⁶ has shown experimentally that the intestine whose serosa shows peritonitis but no distention gives the same motility tracing as the normal bowel and paralysis is not demonstrable.

Inflammation of the peritoneum covering the intestine and of the adjacent intestinal wall results in an interference with the nervous mechanism, producing a paralytic form of ileus with its great meteorism, toxemia, depression, fecal stasis, exhaustion from pain and loss of sleep, finally resulting in fatality.

My own observations lead to the conclusion that several factors enter into the distention; peritoneal toxemia, intra-intestinal toxemia, circulatory disturbance, and gaseous distention. The intestine when incised in such cases tends to contract at least in a limited portion. The entire picture of ballooning of the intestinal coils and distention of the abdomen cause a marked interference with cardiac and respiratory function by mechanical pressure in addition to the toxic depression.

The vomiting, which is an important part of the clinical course of this pathology, has been variously explained. The irritative vomiting occurring early in the disease is probably of reflex origin, while that occurring later is due to toxic paresis, distention and increased fluid within the intestine.

Certain cases of perforative type occur in which the causative agent is less virulent. The course of such cases while severe is less rapid than the septic type. These cases in the first moments after infection show similar pathology to that occurring in the septic type. The changed appearance of the serosa is due to desquamation and degeneration of the endothelium. The surface becomes cloudy in a short time, the result of a transudation which forms a coagulable lymph at points of contact between coils. The serosa is thickened, softened, reddened, injected, and sometimes ecchymotic. It strips readily from the muscularis. The entire intestinal wall becomes softened, friable, and markedly congested. It is easily torn through its entire thickness.

The parietal peritoneum is similarly affected, but not to the same degree. The omentum tends in a large proportion of cases to cover the inflamed area and isolate the healthy from the diseased peritoneum. This process often fails of its purpose and the entire peritoneal surface may be involved. The agglutinations of the intestine in this type are the result of soft deposits of fibrin along lines of contact. These also tend to protect against further extension. When observed in this condition, the abdomen presents marked distention of the intestinal loops. Free

gas may escape when section is made. This is of rare occurrence unless a leak is still patent. In some cases a considerable amount of necrosis is present. The "green groin" of the older writers is occasionally met in cases of appendicular origin. This type of peritonitis tends to progress to a fatal termination, the pathological changes increasing until the end. The terminal changes are similar to the septic type of peritonitis as far as the distention and cardiac depression are concerned. The local inflammatory changes, however, are more marked. The amount of free fluid within the abdomen in the early stages is much greater than in fulminant cases. The quantity and consistence of the fluid are not entirely dependent upon the severity of the disease, but the relationship is close.

In these more acute suppurative processes in which pus pockets form or which fail to drain completely after operation the neighboring mesenteric vessels may become thrombotic in varying degrees. Occasionally the clots become softened from peptonizing action of the bacteria, resulting in the transportation of minute infective fragments to other portions of the body. These emboli form infarcts in the liver, lung, kidney, brain, joints, and bones, which give rise to local inflammatory processes and terminate in abscess formation. This phenomenon is accountable for many of the complications of peritonitis. Pneumonia, pleuritis, meningitis, nephritis, arthritis, osteomyelitis, pyemia, and septicemia are frequently associated with this affection.

The necropsy findings in persons dying of general spreading peritonitis are quite characteristic. The abdomen is markedly distended, the skin being considerably stretched and often mottled in appearance. The whole abdominal wall is rigid. Section through the wall shows some edema and softening. In a few cases sloughing of a portion of the wall is observed. Even in cases not affecting the biliary structures the tissues of the wall may be bile stained. This staining is exaggerated when the affection began in the biliary passages. Inflammation of the pancreas with escape of its secretion is shown by fat necrosis and tendency to the formation of sloughs. Similar changes may be observed in peritonitis following ruptured duodenal or gastric ulcer.

On opening the peritoneum in cases of the perforative type of the affection, free gas escapes from the sac. Free gas is also found in non-perforative cases in which a culture of putrefactive organisms is present. The intestinal walls are deeply injected, greatly distended and may be attenuated in part or thickened, softened, and edematous. They are exceedingly fragile, easily torn and contain a very foul-smelling, irritating fluid material. Pockets of pus of varying size are seen lying between the coils and in the different recesses. Soft, fibrinous material

holds the coils in apposition. This is easily separable. Flakes of coagulable lymph are lying over the lines of contact. The quantity of free fluid varies greatly in different cases. The lymph nodes may or may not be enlarged.

The less acute cases seldom comes to autopsy. The findings in this class may be observed upon the operating table. The process is similar to that previously described in spreading peritonitis, but may be checked at any stage of its course. Mechanical removal of the source of infection either by excision of an infected organ such as a necrotic appendix, gall-bladder or an inflamed tube or ovary, or the closure of a leaking ulcer may check the progress of the disease. The subsequent changes consist in resorption of the exudate, the removal of the fibrinous agglutinous formations and the restoration of the circulation. Those cases which are less acute may subside spontaneously in a similar way.

Certain types are localized processes throughout showing little tendency to spread. This is more particularly true of the milder forms due to tubal inflammation. Such cases only occasionally become spreading in type, following leakage. In such cases the production of fibrin is a prominent change. The intestinal coils become somewhat firmly united by the new plastic material. The omentum firmly covers the entire focus and the process of repair is very active. Such changes evidence the small virulence of the bacteria and the active reparative forces of the individual.

Any type of peritonitis which progresses to recovery tends to leave bands of agglutination. These may appear as delicate membranes or veils, firm fibrous structures or closely fused intestinal coils. Seen subsequently at operation or autopsy from other causes these structures may be found to persist. The greater proportion of them disappear, however, in time. Those which persist tend to result in intestinal stasis and complete ileus by their mechanical action.

Microscopic Changes.—In some of the milder forms of peritonitis the cellular picture is quite definite, but is limited in extent. Minute ecchymoses are scattered through the damaged tissue because of the interference with the circulation. The endothelium of the free surface is lusterless, softened, and the cells are swollen. They take stains less readily than normal. Some of these cells show irregularity in shape and size. Apparently they are undergoing degeneration. A process of desquamation is observed in some of them. In severe cases they are necrotic and fail to take a stain. In the very mild cases mitosis of these cells may be observed. Later in cases in which repair is active the mitotic figures are more frequently seen. On the surface of the serosa

and overlying the endothelial cells, numerous leukocytes are observed. Nuclear change is taking place in these cells and their chromatin substance escapes into a layer of fibrin in which they are enmeshed. Numerous bacteria are observed enclosed within these cells and some are also seen free in the fibrinous exudate. Many macrophages and eosinophiles are seen. Fibroblasts occasionally wander into this exudate and are particularly numerous there in the stage of repair. They actively participate in the production of adhesive material and the formation of permanent adhesions. In this process an active part is also taken by the angioblasts which enter into the production of new vessels. They are observed in active mitosis.

The lymph vessels are considerably dilated and contain fibrin and large numbers of leukocytes. The blood-vessels are distended and choked with cells. Sometimes evidence of complete stasis may be seen. The endothelial cells of both the lymph and blood-vessels are distended. Even in the mildest cases small hemorrhages are observed in the connective tissue spaces. Very severe cases show this occurrence to a marked degree. The serosa is thickened. The subserosa is actively involved. Its connective tissue is edematous, infiltrated with leukocytes and its cells show karyokinesis. The same fibrinous material found upon the free surface is observed in the intercellular spaces of this layer as well as of the serosa. Similar changes may be demonstrated in the muscularis. Usually these changes are less extensive.

Some observers claim that the nerve structures in the walls are edematous, hydropic, show vacuolation, and lose their staining reaction. They also lose their power of transmitting impulses to the centers. These changes account in part for the loss of intestinal tone and the extreme distention.

The amount of plastic deposit observed in some cases of peritonitis is marked and is shown microscopically to be due both to an increase in the number of cells and to edema and swelling of their protoplasm. These masses as formed in the tissues both of the intestine and omentum surrounding inflammatory toxins are readily seen by the naked eye. Such structures disappear promptly following subsidence of the inflammatory process. These thickened masses have been mistaken clinically for malignancy.

The microscopical diagnosis of the inflammatory character of such structures is based upon the presence of an exudate which is not yet organized, upon the large numbers of polymorphonuclear leukocytes showing nuclear destruction and the presence of many plasma cells and phagocytes. The determination by staining methods of bacteria within

the cells is also strong evidence of the inflammatory nature of the process.

The examination of sections made during the late stages of repair following the milder subacute types of peritonitis shows the new tissue to be undergoing organization and more mature types of cells are observed. This is particularly true of the fibroblasts and the angioblasts. In this stage the differentiation of the cells is made with greater accuracy.

The more acute infections of the peritoneum show marked necrosis of cells and an increased amount of ecchymosis. This is the result of the direct toxic effect of the bacteria on the serosa. The evidence of mitosis is less in these cases until the stage of repair is reached.

Chronic serositis, so-called, shows the changes of a chronic inflammatory process with hyaline degeneration of the cells.

So-called carcinomatous peritonitis presents the appearance of a hyaloseritis. This tissue appears under the microscope as thickening of the connective tissue containing small nests or rows of epithelial cells resembling in structure the sections of the primary growth. These structures are edematous and the peritoneal sac contains a quantity of thick albuminous fluid, which reforms promptly upon removal.

SYMPTOMS AND DIAGNOSIS—ACUTE GENERAL PERITONITIS

Notwithstanding the fact that there are so many causes acting in the production of the diffuse type of peritonitis, there are certain symptoms present which are common to all.

There is an intense toxemia present which varies somewhat with the intensity of the infectious agent and also according to the coincident causative affection. In some very fatal cases the symptoms of septic intoxication predominate, and death may take place before the local lesions and their symptoms have time to develop fully (septic peritonitis). In such cases the patient is overwhelmed by an intense toxemia following promptly upon an intra-abdominal calamity. Sudden pain in the abdomen, marked depression, cold clammy sweat, rapid feeble pulse, perhaps a subnormal (but more often a rapidly rising) temperature with general abdominal rigidity and tenderness will distinguish the condition. Vomiting is present in almost every instance. Hiccough soon occurs and shortly death ends the scene.

In other cases the affection develops more slowly, the history giving evidence of some one of the many lesions which are prone to result in this form of peritonitis being present for some time preceding the

onset. Each of these antecedent lesions carries a symptomatology of its own, which will be given under special heads.

The prominent and constant symptoms are abdominal pain and vomiting. The pain is severe, sharp, and constant, and it may follow a period of quiescence, particularly in those cases following appendicitis or after gangrene from ileus. Tenderness and rigidity of the abdominal wall are distinctive symptoms. They are almost always accompanied by abdominal distention. An exception is sometimes seen in perforation from a typhoid ulcer, where the abdomen may be scaphoid in appearance at first, later the meteorism is marked. The patient looks very ill. The facial expression is anxious, the eyes are sunken, the features pinched, and cyanosis is soon noticed. The mentality may be clear throughout, but frequently it is dulled before death. The pulse is usually rapid, the rate increasing and the volume small. The respiration is shallow and increased in frequency, apparently in the effort to avoid pain. As the abdominal distention increases the respiration becomes more superficial and in the final hours it is labored. From the collection of mucus in the bronchi harsh respiratory sounds are audible. The cardiac action becomes markedly impaired because of abdominal pressure, as well as from loss of strength due to the intense toxemia. At the same time the blood-pressure becomes diminished and the pulse may scarcely be counted. Leukocytosis is present and the polymorphonuclears are relatively increased, strong evidence of a suppurative process.

The bodily temperature is increased, with the exception of a sudden fall which sometimes occurs following the causative intra-abdominal calamity. It should be borne in mind that the temperature curve is no exact indication to the gravity of the disease. Even in those cases where the axillary temperature is low, if taken in the rectum it may register several degrees above normal. This is undoubtedly due to the local heat of the inflammatory process within the abdomen.

Anorexia is absolute during the activity of the process and is the result of the highly toxic material in the alimentary canal. No food products can be assimilated under these conditions and their administration before the subsidence of the vomiting and the distention shows poor judgment. Solids or fluids taken into the stomach are promptly rejected. The return of a desire for food is always a sign of amelioration of the symptoms. Thirst is pronounced because of the dehydration from vomiting and perspiration.

The Urine.—The quantity of this secretion is diminished and the color is high. Indican is usually present in considerable amount. It is not unusual to find a small amount of albumin and in some instances

casts are observed. The bladder function may be obtunded, but in some cases micturition is frequent and painful.

Pain in Peritonitis.—The occurrence of abdominal pain is one of the most constant and earliest symptoms. It may be localized early in the disease to the region first involved. Soon it becomes general over the abdomen and is accompanied by corresponding tenderness. This tenderness is increased on pressure, and is a diagnostic sign.

It is very important that particular notice be taken of the initial pain as well as of later localized pain, since this usually leads to a diagnosis of the causative condition.

When the cardinal symptoms are present the diagnosis is easy, yet in many instances it is not made.

There are so many conditions which simulate the symptoms of general peritonitis and so many other intra-abdominal conditions terminate in peritonitis that the most careful study of each case must be made in order to avoid error. Most of the errors in diagnosis result from a lack of care and study of the case, rather than an actual lack of knowledge. Careful observations, careful history taking, the ability to focus the facts make for competent diagnosis. The one who can best evaluate the symptoms, lay aside the unimportant and direct his attention to the important ones rarely fails to arrive at a correct conclusion. He should have confidence in his ability to reach a correct conclusion, and having formed an opinion should not hesitate to express it and stand or fall by it.

The symptoms of acute peritonitis are usually sudden in onset, sometimes following promptly upon the reception of a blow upon the abdomen, which may produce rupture of a hollow viscus without any external evidence of damage.

It may follow very closely upon the symptoms of acute appendicitis, an acute strangulation of the gut from volvulus, internal hernia, strangulation from a band, a Meckel's diverticulum, an external strangulated hernia, suppurative or gangrenous (phlegmonous) cholecystitis, acute pancreatitis, rupture of a gastric or duodenal ulcer, knife or gunshot wound, rupture of an hepatic abscess, a parietal abscess, leaking suppurative salpingitis, acute gonococcic infection, acute puerperal sepsis from labor at term, or from abortion. Because of these facts the previous history must be taken with great care because each of the preceding diseases has symptoms of its own. All may have something in common.

After a sudden onset or a very brief illness spreading peritonitis always presents pain, general tenderness, and general abdominal rigidity. The abdomen often becomes "boardy" hard. Tympany is present

throughout, except where a circumscribed abscess or an intussusception lies, when dulness may be elicited.

The presence of tympany over area of normal liver dulness means a perforation at some point in the alimentary tract. Vomiting is a constant symptom.

Borborygmus, which is present early in a number of causative lesions, soon gives way to intestinal paresis due to the inflammation of the intestine. In many cases no gas or feces pass.

The presence of the above symptoms makes the diagnosis of general peritonitis positive. Careful attention to the development of the symptoms presenting will in most cases also differentiate the accompanying disease.

To distinguish the causative condition prior to operation is a more difficult matter. This is accomplished by careful attention to the history of the onset and the localizing symptoms.

Conditions which may simulate beginning peritonitis are:

Intestinal colic. The pain is always intermittent, varying in intensity. Pressure on the abdomen does not elicit tenderness; firm pressure relieves the pain. There is no abdominal rigidity. Temperature and pulse are usually normal. Vomiting may be absent or in any event is not a marked symptom.

Dietl's crises, first described by von Dietl, of Vienna, are the results of a kinking upon itself of the ureter. Such an attack is evidenced by severe pain, chill, nausea, and vomiting. The pain follows the course of the ureter. The surface is cool and covered with sweat. There is no elevation of temperature, no general abdominal tenderness or rigidity.

Tabetic crises. The sudden severe intra-abdominal pains which occur in locomotor ataxia, particularly at night, have been mistaken for acute peritonitis, but a careful examination should readily exclude the latter condition.

Acute gastro-enteritis. The characteristic symptoms in this condition are vomiting and diarrhea. The latter is usually absent in acute peritonitis, but may be present at the onset. It soon ceases when peritonitis becomes established. There is general abdominal soreness in gastro-enteritis, more of a diffused tenderness than actual pain. Abdominal rigidity is absent. Occasionally the leukocyte count may be increased, but the ratio of polymorphonuclears is unchanged.

Nephritic colic sometimes simulates very closely the onset of acute peritonitis. Nausea, vomiting, and abdominal distention may be present. Some reflex rigidity may also be observed. The temperature is normal and the pulse not materially accelerated. A complaint of general ab-

dominal pain may be made. There is always pain over one or both kidneys. The pain radiates from the kidney to the testicles and bladder. Retraction of the testis may be observed. Anuria may be present. Catheterization may obtain sufficient urine for examination. The presence of crystals and blood will be sufficient to excite strong suspicion of gravel. This train of symptoms makes the diagnosis of renal colic justifiable. A few hours will render the diagnosis positive.

Uremia is sometimes associated with abdominal distention and severe pain. The absence of rigidity, marked general or localized tenderness and with the presence of blood and casts in the urine will usually make the diagnosis of this condition. Suppression of urine is not infrequent. The mentality is often benumbed and coma or convulsions may be present.

Gastric ulcer. History of repeated attacks of gastric disturbance, pain and vomiting after eating, sour stomach, belching, eructations, localized or circumscribed tenderness with pain extending to the back, absence of fever, presence of blood in the vomitus on the start will usually make the diagnosis. Impending or actual perforation is evidenced by sudden sharp localized pain, soon becoming general over the abdomen, with boardlike rigidity, nausea, and vomiting, with the history of ulcer. Fever and increased leukocytosis appear as peritonitis is set up. The rigidity becomes general and marked.

Acute cholecystitis usually gives due notice of its presence before leakage occurs, and if under observation prior to this event a rounded mass with localized tenderness is observed. The temperature is usually higher than in the other causative conditions mentioned. Jaundice is a frequent symptom. An icteric tinge to the conjunctiva is a most valuable pointer to the source of the trouble. When rupture takes place severe pain in the right hypochondrium is observed, vomiting is present and very painful.

Gall-stone Disease.—Indigestion for some time, intermittent attacks of pain some time after meals, or frequently at two or three A. M., with nausea and vomiting and the presence of muddy sclera or skin, point to gall-stones. The presence of fever, a tender mass in the right upper quadrant mean an acute cholecystitis. Sudden diminution of the size of such mass followed by shock, great pain, general abdominal tenderness mean a gall-bladder leak and onset of peritonitis.

Intestinal Obstruction.—The various forms of this affection are evidenced by sudden sharp and persistent intra-abdominal pain, absolute obstipation, great distention, and marked evidence of distress. There may be a history of some preceding interference with fecal flow or not.

Usually there is no evidence of preceding illness. In those cases due to intussusception, bloody stools and mucus may pass, but there is no passage of gas. In cases due to malignant disease there may be a history of previous attacks of diarrhea or constipation. Frequently these have been absent. There is no elevation of temperature, and the pulse rate is not accelerated.

This symptom complex points to intestinal obstruction. One of the prominent diagnostic points is the presence of visible or palpable waves of peristalsis. Peristalsis soon ceases in the presence of peritonitis. The distention in obstruction may be localized to one portion of the abdomen. The rigidity is usually less marked and the pain is more localized to the site of obstruction.

Intussusception occurs most frequently in children, in whom its onset is very sudden. The sudden onset of intra-abdominal pain in a healthy looking child under ten, accompanied by passages of bloody mucus but no feces or gas, particularly with a doughy tumor, means intussusception.

In the adult a similar occurrence may be preceded by a previous history which is suggestive of the possibility of an intestinal tumor. When such symptoms arise, followed in a short time by the usual symptoms of peritonitis, the correct diagnosis should follow.

In volvulus the pain is central, very severe with spasmodic exacerbations, and is accompanied by very active peristaltic waves. In this condition the general abdominal pain appears after some hours.

The pain in appendicitis is usually first noticed at the umbilicus and accompanied by nausea and vomiting. Very sudden in its appearance it soon is located in the right lower quadrant and the site of greatest tenderness is over McBurney's point, about midway between the anterior iliac spine and the umbilicus. This site is not constant and in cases where the appendix lies in the pelvis the patient complains of pain on the left side. The point of greatest tenderness is immediately over the base of the appendix as a rule. Sudden cessation of the early pain indicates rupture of the appendix and is very misleading. Sometimes soon after rupture has occurred, very little tenderness may be elicited. Very rarely rigidity is found to be slight.

When rupture of an appendicular abscess has taken place and in some cases following rupture of a necrotic appendix the pain ceases suddenly, followed promptly by a very sharp pain and great shock. This indicates a calamity and calls for immediate intervention. The writer has observed a very rapid and progressive rise of temperature in a few instances. The temperature, however, may fall following such rupture, and is no positive indication of the gravity of the condition.

Perforation for typhoid fever is usually preceded by a number of days' illness, with strongly presumptive evidence of this condition or a diagnosis if the patient has been under observation. Occasionally, however, a patient may come into the wards with mentality obtunded and no history of previous illness obtainable prior to a sudden pain in the right iliac region. This occurrence makes the diagnosis more difficult. Such a case came into the Louisville City Hospital, and the correct diagnosis was made based upon the following facts. The temperature was subnormal, the abdomen scaphoid. After a search the spots of Louis were observed and the leukocyte count was 9600, the urine normal. This case is recited to call attention to the possibility of error.

Perforation of a gastric or duodenal ulcer is almost always secondary to some symptoms of such affection. The pain is sudden, severe, and localized at the upper portion of the abdomen. It occurs near the pylorus and remains for some time a right-sided affair. Eventually the pain and tenderness become general. The temperature is frequently lowered at first and the leukocyte count is not so high as in appendicitis during the early hours. The amount of shock is considerable. The boardlike resistance in the epigastrium is marked.

GENERAL OBSERVATIONS ON PROGNOSIS IN PERITONITIS

The important factors for consideration in this connection are:

1. The type of the disease, whether localized, spreading, or general.
2. The source of contamination.
3. The bacterial flora active in the production of the infection.
4. The resistance of the patient to this form of disease.
5. The use of purgatives.

Under the first heading the surgeon determines from the sharpness of onset, from the severity of the pain, and from the general reaction something of the acuity of the process. He soon becomes able to determine whether he is dealing with a mild local lesion or whether a localized or a spreading peritonitis exists. One of the best examples is a case of appendicitis; which may pass through all three stages. It may begin as a mild endo-appendicitis with subsidence of the symptoms in a few hours. It may progress to the point of distention with inflammatory fluids and result in a localized peritonitis with capping of the inflamed appendix by a fold of the omentum or layers of the gut, or it may continue until diffuse peritonitis sets in. Should the inflammation progress, an abscess forms about the appendix within the mesen-

teric and omental folds which have become united as a result of the local peritonitis.

When such an abscess forms as the result and because of necrosis and rupture of the appendix, there is in nearly every instance a sudden relief of pain. This relief comes as a direct result of the relief of tension within the walls of the appendix. This sudden cessation of pain is of great value both in diagnosis of the exact lesion present and in giving a correct forecast of the outcome of the case.

The surgeon should recognize this as a localized appendicular abscess with necrosis of the appendix and localized peritonitis, which may go

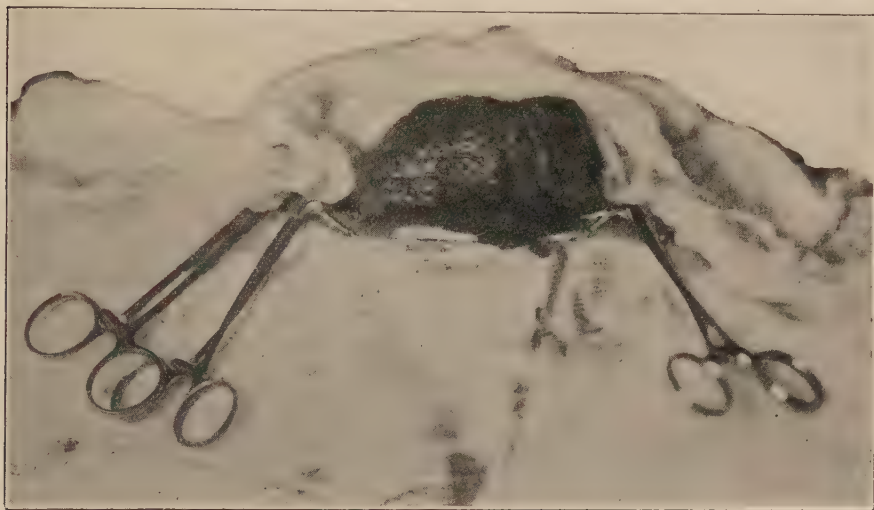


FIG. 33.—OMENTUM CAPPING APPENDICULAR ABSCESS.

Note: This abscess after gauze had been placed around burst with a noise like the rupture of an over-distended rubber balloon from gas in abscess sac.

on to a doubtful recovery without surgical intervention, but which will give a remarkably low mortality if it comes to immediate operation.

If a patient is seen later in the disease when the sharp primary and early pain has suddenly subsided or disappeared entirely leaving some persistent tenderness, perhaps a palpable mass, followed by a gradual or sudden increase in the pain, more local or perhaps general abdominal tenderness, the diagnosis of a leaking appendiceal abscess with beginning general peritonitis is permissible. The prognosis under these conditions becomes very grave, and further delay in operation is not considered unless the patient soon after the rupture rapidly goes into shock and becomes moribund, which makes operation unadvisable.

The prognosis in these cases of ruptured appendiceal abscess becomes

momentarily more grave. In evaluating the outcome the surgeon must consider the general appearance of the patient, the facial expression, the amount of pallor, the dryness or moisture of the skin, the character, rate, compressibility, tension of the pulse, the respiration, as well as the extent of abdominal rigidity. In addition the leukocyte count accurately made is of great value in determining the patient's resistance. A high leukocyte count always tells that the patient's army of defense is on the field. Per contra, a leukopenia under these circumstances always means a low resistance, and usually also means a violent infection. The temperature in such a case as described is but a poor index of the outcome. The sudden cessation of pain has misled the attendant very often in cases of appendicitis with localized abscess. Sometimes, too, the pulse is also deceptive, being normal in rate and volume in the presence of grave local lesions.

A number of years ago one of my surgical colleagues asked me to see a physician with him who had been ill for about a week with appendicitis, and who was insisting because of the absence of elevation of temperature or increase in the pulse rate that he was well and should be permitted to go home. His surgical attendant explained to him the dangers incident upon his affection, and insisted upon his recumbency and perfect quietude, expecting to open the abdomen the following day, in which opinion I concurred. A few hours later this patient got out of bed, his abscess ruptured, and he went rapidly into collapse, dying a few hours later. This case exemplifies the fact that the prognosis, in appendicitis or any other lesion in which peritonitis may develop, should be guarded. Such lesions are always serious. Experienced surgeons appreciate the gravity of acute general spreading peritonitis, and at sight can readily recognize impending dissolution.

The pale countenance, the moist, clammy skin with beginning lividity of the nails, coldness of the extremities, pinched *alæ nasæ*, compression about the lips, the sardonic grin, difficult respiration, staring, wide open eyes, persistent vomiting, distended abdomen, hiccough, are signs which mark the approaching end. The patient may be mentally alert, but often drifts into lethargy or coma. The surgical student should be impressed with the fact that a correct prognosis is of as much importance as a correct diagnosis. There is no escape from an error in prognosis and to the family and friends this is really the important fact. In arriving at a correct opinion of the outcome of a case, the great variation in the severity in different cases of peritonitis must be borne in mind.

The source of the contamination, when determined, will prove of value in estimating the outcome. The danger varies from the simpler

forms of traumatism with but a slight infection and prompt local repair, to the most severe cases of perforative or of puerperal spreading processes, and the prognosis should be given accordingly.

The study of the bacterial flora active in the causation is very important, and in institutions where laboratory studies can be properly carried out and recorded, the findings will be of value in arriving at a proper prognosis. A long series of cases is necessary to make the findings worth while from a scientific standpoint. Such careful studies may cause considerable modification in the views held at present.

The present belief is that the most violent cases will often show a mixed infection. The streptococcus (viridans and hemolyticus) is probably the most virulent type of organisms causative of this disease. Some of the cases which follow the blood stream infection and which are met in connection with suppurative pleurisy and pericarditis show this form of organism. In some of the studies made during the war in the influenza epidemic, this type of infection was often found superimposed upon the influenza bacillus and pneumococcus infections. In the larger number of cases of pleurisy where such mixed infection occurred, the streptococcus overgrew the other organisms present. It is, therefore, probably true that such overgrowth occurs in peritoneal infections.

The staphylococcus is a very hardy organism, and at times assumes an extremely virulent form.

The *Bacillus coli communis* is a normal inhabitant of the alimentary tract, particularly the colon. It is, therefore, often the sole bacterial cause of appendicitis and of peritonitis. This is one of the most frequent of the contaminating bacteria in cases where more than one variety is present. In some cases it shows a gas producing property, and this may excite suspicion of the presence of *Bacillus welchi*. *Bacillus coli* was often seen in the cases of "green groin" so frequently met in the old days, but which are rarely seen at present, since appendicitis cases nearly always come to operation early.

The *Bacillus pyocyaneus* is also a frequent contaminating organism, if not a frequent causative one. Usually the process is not grave, but this organism is very viable and the green pus may persist through a long and tedious convalescence, where an ugly abscess has been drained and hot packs employed.

Peritonitis arising from gonococcic infection or from the tubercle bacillus, each has a prognosis of its own.

It is not always possible to estimate exactly the resistance of a patient to infection. Much will depend upon the general vigor, the

physique, the nourishment of the individual. Pale, pasty, delicate, poorly nourished individuals make poor surgical risks. Intercurrent disease, as pneumonia, pleurisy, tuberculosis, Bright's disease, diabetes, cardiac disease, adds much to the gravity of a given case.

A study of the circulation and respiratory function and of the general condition, taken in connection with the blood picture, will usually lead to a correct estimate. Children are, as a rule, less able to withstand attacks of appendicitis and peritonitis than adults. This seems in part to be due to the amount of lymphoid tissue in the appendix, which lessens in later life. The absorbent vessels are particularly active in children, and there seems to be present less resistance to bacterial invasion. The prognosis may be made less favorable, too, by the fact that children may not carry the complaint of pain promptly to some older member of the family, or the pain may be attributed to something ingested. In this way the child may not come under observation as early as the adult.

Purgatives.—It seems very clearly demonstrated that the use of purgatives by the parents or in some cases by the physician before a case of this kind comes to operation, adds very greatly to the dangers. In the hands of most observers the mortality rate shows a distinct increase in cases which have been purged. Also in cases which do not result fatally, the convalescence is more stormy.

NONOPERATIVE TREATMENT

In no department of medicine is more mature judgment demanded of the surgeon than in the care of this condition. Just as the ability to apply the proper treatment to a case of typhoid fever in the old days went a long way in the preparation of a physician to handle other medical conditions, so does proper instruction in the care and treatment of this affection fit the young surgeon for the treatment of other serious surgical lesions.

About the treatment of acute general peritonitis lies a large portion of surgical progress during the past half century. The literature abounds with contributions upon this subject, all of which possess some value, some of which is epoch-making. It is impossible to go into detail and some valuable reports will of necessity be omitted here.

The names of Alonzo Clark, Reginald Fitz, W. T. Bull, Deaver, McBurney, Joseph Price, John B. Murphy, Ochsner, Robert Morris, McMurty, Blake, Finney and John G. Clark, illuminate the literature upon this subject in this country, while those of Lister, Tait, Treves,

Bland-Sutton, Moynihan, in England, Mikulicz and Kroenlein, in Germany, have been important contributors.

There has been a remarkable change in medical and surgical opinion upon the management of this affection during this period, largely due to improved surgical methods.

Medical Treatment.—The dogma of some that there is no medical treatment is not tenable. Many cases of peritonitis are prevented by proper medical care, while many cases are exaggerated by improperly applied efforts at medical management, such as the use of purgatives, the injudicious use of opiates, and the ill-advised employment of manipulations. It is not well for surgeons to become too “cock sure,” and sound medical opinion is to be sought, not disregarded.

In the early work in the treatment of this condition, when surgical intervention was extremely serious, Alonzo Clark's work stands out. He was honest in his convictions, and as daring in their execution as the boldest surgeon. He conceived the idea that rest was the desired thing in the treatment of peritonitis and proceeded to obtain it for these patients by “heroic doses” (his own words) of opium. For days he kept the sufferer narcotized with increasing doses of opium, until a maximum of 32 grains of morphin, or 261 grains of opium were administered in twenty-four hours. No one would dare give so much at this time. He obtained much improved results, and his plan was followed generally, but with less intensity.

Opium has its place in the treatment now, but its part is small. It is unwise to mask symptoms prior to diagnosis by its use. After diagnosis is made, it is of value to relieve acute intra-abdominal pain, to support the patient pending operation or through his journey to the hospital. It is excellent given with atropin to overcome shock since it obtunds sensibility, quiets the patient, does away with restlessness, brings warmth to the skin, and strengthens the pulse.

Following upon the massive doses of opium used by Clark and his followers in the management of acute peritonitis came the Tait school. Lawson Tait held the opinion that there was much benefit to be derived from the proper use of saline purgatives in the treatment of postoperative and other forms of acute, as well as localized peritonitis, and this method was somewhat widely employed in the early nineties. It was supposed to cause some depletion of the inflamed structures, and at the same time to remove a large amount of noxious material from the alimentary canal. Some benefit was obtained in a considerable number of cases by this plan of treatment. It later developed, particularly following the work of Ochsner and Murphy, that in many cases this

plan of procedure was harmful, since there resulted an increased tendency to spread the infection. Moreover, catharsis often produced marked depression in these cases, and occasionally when the gut was parietic and where partial ileus was present, the results were disastrous.

Subsequent to the discussion of this subject at the Birmingham meeting of the Southern Surgical Association, this plan of treatment was abandoned.

It has been found that the intensely toxic material within the intestine in cases of general peritonitis, in which there is an active bacterial growth, may be very safely removed by repeated gastric lavage. This method may be carried out without much distress to the patient as soon as the dread of swallowing the tube is overcome. In the writer's opinion it is one of the most important life-saving measures in our armamentarium in the treatment of this disease, both before and after surgical operation. Many surgeons wash the stomach out prior to anesthesia, claiming it lessens the danger of drowning from the inspiration of vomitus. Others are content to employ it during or after anesthesia, both in peritonitis and in ileus. It prevents nausea, lessens pain and discomfort, lessens distention, improves the tone of the gastro-intestinal tract. The danger incident to its use is very slight. It is contraindicated in perforation from gastric and duodenal ulcer and that of typhoid fever until after suture of the leaking organ is accomplished.

Ochsner's Plan.—To Ochsner must be given the credit of first recognizing the importance, in extreme cases of acute peritonitis with marked depression, hanging onto life by a thread, of the starvation method to bring the patient who is inoperable to an operable condition. By withholding food, providing rest, giving the patient time to rally, and by the employment of stimulants by hypodermic method, he concluded these almost moribund cases which the additional tax of an operation would almost certainly kill, could be safely brought to operation and a happy outcome. Add to this the judicious use of the Murphy drip method of proctoclysis, and a very happy combination results.

There was a storm of disapproval of Ochsner's report, particularly from the advocates of immediate abdominal section in perforative peritonitis, and particularly of that form due to appendicitis, based largely upon their failure to recognize Ochsner's position. Ochsner believed in and taught the value of early operation in appendicitis, but deprecated operation in those cases where the tax of intervention would throw the balance against recovery, and believed such patients in many instances could be brought to the point where a section would be safe.

Proctoclysis.—The employment of the instillation of fluid into the rectum in the management of acute peritonitis and also of acute appendicitis is one of the many valuable contributions of John B. Murphy to surgical art.

Following acute abdominal inflammations and particularly following serious surgical procedures for their relief, there is often observed marked dehydration of the tissues. This is evidenced by the extreme thirst so often present. Accompanying this condition there is in many cases an intense toxemia present and an accompanying acidosis as well. These individuals are unable to retain fluids in the stomach, vomiting being easily excited, often annoying and painful.

This state of affairs is much ameliorated by the use of rectal instillation of normal saline as recommended by Murphy. The rate of flow is controlled so that from 40 to 60 drops are delivered from the tube per minute. Some observers claim equally as good results from the use of tap water as from the saline solution. My personal experience favors the latter. It is often well to add one dram of sodium bicarbonate to the pint or 1 or 2 drams of glucose in cases of acidosis. The addition of 15 minims (1 c.c.) of adrenalin solution 1:1000 is valuable in cases of depression to carry the excess load over a temporary period of exhaustion.

Some surgeons have attempted to substitute the use of the subcutaneous instillation of saline solution for the Murphy method, but with the exception of its use for immediate resuscitation purposes after operation it is less satisfactory. It fails entirely to obtain one of the most advantageous functions of the rectal method, which consists in the dilution of the intra-abdominal toxins and the lessening of the rapidity with which they are taken up by the absorbent vessels.

The patient becomes water-logged much earlier by the subcutaneous method.

George Ryerson Fowler, unfortunately a victim of the affection which he did so much to relieve, advised the sitting posture in the bed for this class of cases, believing thus to delay absorption and permit the full development of the protective forces of the individual.

Results from the combination of the two plans of treatment used in conjunction have been uniformly satisfactory.

SURGICAL TREATMENT

Preventive Treatment.—This is a very important part of the treatment of acute peritonitis. Its scope is also very wide, but as the

causative factors are covered under separate headings it will only be necessary to deal with it briefly.

First in importance in the prevention of peritonitis is to keep all abdominal wounds clean and to prevent soiling of the peritoneal sac during the necessary operative manipulations. The most perfect technic is necessary to accomplish such result.

One of the most frequent causes of peritonitis is inflammation of the vermiform appendix. A complete knowledge of this subject is necessary so that a prompt diagnosis may be made before contamination of the peritoneum has occurred.

A most important point in the prevention of peritonitis from this source is refusal to administer a purgative to any patient suffering from intra-abdominal pain. There is nothing connected with this affection that increases the mortality in appendicitis as much as the use of purgatives. By increasing peristalsis it spreads infection, excites nausea, and increases pain.

Upon the diagnosis of appendicitis, operative measures should be instituted at once. In those rare cases too ill to withstand operation or where something prevents its employment, food and water by mouth should be withheld. The patient should be put to bed in Fowler's position and an icebag applied. Heat is permissible as a substitute for cold. Manual manipulations are positively contraindicated. By lessening the inflammatory process by these measures an attack of peritonitis may be prevented.

The usual measures of prevention of puerperal contamination as described elsewhere will prevent peritonitis from this source.

The prevention of gonorrheal infection is essentially important in prophylaxis. These measures are generally understood and if carried out with vigor much may be done to accomplish the reduction in the frequency of peritonitis of this type. Such patients should be placed in bed and treated locally in such a way that the infection will be overcome.

Early relief of hernial protrusions by operation tends to lessen strangulation and incidentally peritonitis.

Prompt operation in ileus, invagination, volvulus, omental or other torsions, mesenteric thrombosis, distention, and gangrenous inflammation of the gall-bladder will lessen the number of cases of peritonitis very materially. Constant watchfulness is necessary to the prevention of these types of infection.

The early operative treatment of gastric and duodenal ulcers which fail of relief by a brief period of rest, proper diet and medication, pre-

vents rupture of such lesions, a prolific source of peritoneal contamination.

Operative Treatment.—The frequency with which the abdomen is opened by the surgeon makes the protection of the peritoneum from contamination of essential importance. In the preparation for an abdominal operation the most meticulous care to provide perfect asepsis is to be observed. By following careful technic in clean cases the development of postoperative peritonitis can be made almost infinitesimal. In cases where operation is done in the presence of pus or where the alimentary canal or the genital tract must be opened, even greater care, if possible, must be used. Sterile gauze or towels must isolate and protect the peritoneum from contamination. Postoperative peritonitis



FIG. 34.—SUCTION TIP FOR ASPIRATION OF THE FLUIDS FROM THE ABDOMINAL CAVITY.

Devised by Dr. E. H. Pool and described in *Annals of Surgery*, 1913, 58: 537.

shows some slackness in the operative technic even in such cases, but it must be remembered that it cannot be entirely prevented in some instances, even by the greatest of care.

The next class of cases to be considered from the standpoint of prevention is that resulting from traumatism. These differ from elective operative cases since the wounds are made without any surgical or even ordinary cleanliness, and are in that particular much more prone to be contaminated. It will be the purpose, therefore, of the surgeon to prevent the further entrance of microorganisms into the cavity and render innocuous those already present.

He should bear in mind that the sooner treatment is instituted in perforating or even penetrating wounds of the abdomen, the better the results which will ensue, other things being equal. In treating such cases all penetrating wounds, after proper preparation of the field, should be enlarged for examination to determine the presence or absence of

any visceral injury. As soon as the abdomen is opened the healthy peritoneum should be protected from contamination by the escaping intestinal contents by large packs of wet gauze. The gross contamination of the structures adjacent to the wounded viscus should be rapidly removed by gauze sponging which takes up blood clots and fecal matter. The use of a suction pump is of great value. Foreign bodies



FIG. 35.—ILLUSTRATING AUTOMATIC PUMP OF DR. E. H. POOL, PREVIOUSLY DESCRIBED.

carrying infective material should be removed when feasible, but no time should be lost in needless search since the peritoneum will take care of a foreign body with slight contamination better than the patient will of slow and damaging surgery. Closure of leaking wounds or those which are potential for later contamination should be completed at once, as soon as hemorrhage, the most imminent single danger, is controlled. In some instances where many perforations of the intestine

are found within a limited extent of the canal, a rapid excision is a time-saving and life-saving measure. In no department is good surgical judgment, operative skill, and dexterity more important than here. Insult to the tissues, slow and prolonged operation, tend to increase mortality very materially. Surgical experience and judgment will determine whether the abdominal wound shall be closed with or without drainage.

The next class of cases to be considered from the standpoint of prophylaxis embraces a large group of cases in which the rupture of an abscess, or of the stomach, intestine, appendix, gall-bladder, uterus, fallopian tubes, or the urinary bladder is imminent or has actually occurred. Such conditions present the phenomena of shock, and an abdominal catastrophe presents. The prevention of a general, spreading peritonitis can only be secured by prompt action along the lines presented above. It is certain, however, that in many instances the first desideratum is to overcome shock, and this holds good in perforating wounds as well. Nothing is as productive of shock as hemorrhage, and this must be overcome and the patient's resistance evaluated as nearly as possible before any operative steps are commenced. Practically the same measures as advised in wounds should be employed here and also in cases of internal or external strangulation. Upon the thoroughness in the application of these basic principles of prevention of contamination of clean tissue will depend the success of surgical treatment.

Because of the fact that the majority of cases remain localized throughout and because even the spreading types are local at their inception in most instances this type will be taken up first, while general perforative peritonitis will receive special study.

The management of peritonitis must of necessity vary with the organ involved, with its severity and its extent, for, after all, general peritonitis is only an exaggeration of the local form, in virulence, rapidity, and extent.

Certain conditions which may result in general peritonitis may be conducted safely for longer periods than others. Abscess of liver, cholecystitis, mild forms of salpingitis, duodenal and gastric ulcer may be under observation for considerable time before the onset of symptoms of peritonitis. In any of these cases, however, there may be sudden, violent symptoms indicating the beginning of spreading peritonitis. Should the patient's general condition permit, immediate abdominal section is then indicated. The onset of appendicitis, ruptured gastric or duodenal ulcer, perforation of typhoid ulcer, rupture or gangrene of the gall-bladder, intestinal obstruction from kinks, bands, volvulus, hernia, in fact any abdominal calamity, calls for prompt interference.

Certain cases, as for instance peritonitis due to acute gonococcic salpingitis or even certain forms of puerperal peritonitis, may be more safely handled by delay. Upon this point there is considerable difference of surgical opinion. My experience leads me to believe that in such cases in which the patient is well nourished and no blood stream infection is present, the most satisfactory plan of treatment is by prompt abdominal section. The mortality will not be materially increased, and the economic saving in time necessary for restoration to health will be very great. Cases seen some time after the inception in these forms of the affection are carried more safely through the illness and subsequent operation by delay. It is very doubtful if this holds in the more acute lesions such as appendicitis, typhoid perforation or perforative ulcer of the stomach or duodenum.

There is in this group, however, a limited number of cases which are practically certain to die if immediate operation is done, and which may be brought to the bounds of safety by delay and the application of the method of treatment advised by Ochsner years ago for severe cases of appendicitis almost moribund. This consists in withholding food and even water from the stomach until nausea has entirely ceased, propping the patient up in the Fowler-Rehn position, the employment of normal saline by the Murphy drip method. The use of ice over the abdomen is found most valuable. In addition, gastric lavage until the fluid removed comes clear makes the most satisfactory method of carrying these patients through to the point where operation may be safely undertaken. There is a belief in the minds of some that all the toxic material which prostrates these patients and rapidly brings them to death's door, lies within the abdominal cavity. Our experience leads to the conclusion that the material within the intestine is equally as noxious as the peritoneal fluid. In any event, by gastric and colonic lavage and sewage all intestinal flora are diminished and the absorptive powers increased so that the intraperitoneal toxins can be taken into the intestine and there diluted before being taken into the blood stream. Morphin should be used with careful discrimination, the object being to allay peristalsis, to produce sleep and rest without narcotizing the patient and obtunding the signaling system, of which pain is the important annunciator.

After the reaction from the teachings of Alonzo Clark to the use of Epsom salts, many observers employed purgatives in acute peritonitis, and in acute appendicitis as well. There now seems to be no difference in opinion among the leaders of the surgical profession that purgation in acute appendicitis and in acute peritonitis adds very greatly to the

mortality. The motto should certainly be, "Do not purge"; and the same rule holds good after operation as before. It is much better to wait for the bowel movement for several days and begin with enemata, being sure of the patency of the canal before even a laxative is employed.

After operation practically the same plan is followed. The greatest benefit seems to be obtained by repeated washings of the stomach. That distressing picture of a greatly distended abdomen, persistent vomiting, marked prostration, rapid feeble pulse, shallow respiration, and the expectation of dissolution, can be averted by no medicinal means as readily as by those frequent washings. The distention subsides, the nausea and vomiting cease, the patient goes into a natural slumber, and in many cases to recovery. Where obstructive ileus or paralytic ileus is present with no passage of gas, enterostomy under local anesthetic may result satisfactorily.

In many of the cases of localized peritonitis after operation is completed, very little in the way of after-treatment is necessary. If the patient has been dealing well with the local inflammatory process, he is more likely to be able to handle it after the local source of contamination is removed. His protective forces are developed to their highest capability, and all that is necessary under the circumstances is to let him go on to recovery. Surgical curiosity and meddlesomeness have often made a convalescence stormy when it should have been uninterrupted.

Flushing of the Peritoneal Cavity during Operation.—The instillation of fluids into the abdominal cavity at operation for general peritonitis with the view of removing poisonous products was very much in vogue some years ago.

I have seen many substances employed either to flush out the cavity in the vain hope of ridding it of its pus and load of bacteria, or to kill the poison present by bactericidal action or with the hope of preventing the reformation of adhesions when the primary operation was done for the relief of this condition following peritonitis.

In our very early experience flushing of the abdomen with sterile water or saline solution was regularly employed. It was believed that this was beneficial. Some observers about that time found that if milk was poured into the peritoneal cavity it could not possibly be washed out. The conclusion was reached that it was impossible to remove entirely noxious substances from the cavity. A certain amount would of necessity remain. To take care of the infectious substance dependence must be placed upon the recuperative power of the peritoneum.

Then John B. Murphy pointed out the fact that rapid operation,

closure with drainage, leaving off the flushing, showed such a remarkable decrease in the mortality in acute spreading cases that irrigation was abandoned.

These results seem to show that while flushing was believed to be helpful, it was really harmful, often spreading the infection. The use of bichlorid solutions within the abdomen was soon abolished. Prior even to the above, iodoform was employed in tuberculous cases and abandoned.

The same is true of the use of peroxid of hydrogen in tuberculous peritonitis. This substance has been used to favor control of oozing. We have not used it for twenty years.

Then came Johnson and Crisler with the use of iodine in the acute cases. It left its mark in the large number of secondary operations for postoperative adhesions.

Finally came the use of alcohol in dilution and more rarely the use of ether. The better of these is alcohol if one must use either of the drugs. The advocates of the use of ether claim that the injection of ether into the peritoneal cavity increases the resistance of the peritoneum, also that it favors deep restorative sleep by causing anesthesia of the parts, disinfects the peritoneum and the pus, and transforms the sterilized exudate into an autogenous and specific serum.

We can find no logical reason for the use of ether within the peritoneal sac in the treatment of peritonitis, and this seems to be the general professional opinion concerning its use.

Dakin's solution found some advocates. This substance is so destructive to the peritoneum because of its peptonizing action that it should not be employed.

We are very strongly of the opinion that the cases do best when the leak in the intestine is closed and excess fluid rapidly removed by aspiration. Prompt closure without drainage when we may, and its use when we must, takes care of the situation.

Clinicians long ago reached the conclusion that the instillation of fluids into the peritoneal cavity was harmful. In many of these cases following the peritoneal flushing there developed a very rapid toxemia. This plan of treatment because of such results was abandoned.

It is particularly interesting to note that Peiser⁸ some years later arrived at a similar conclusion in an experimental way. He showed that bacteria were absorbed in quantity from the peritoneum only in the first stage of peritonitis. Subsequently absorption is much slower. Even in the cases of peritoneal sepsis absorption becomes limited, and any bacterial increase which occurs takes place in the blood.

The peritoneum absorbs solutions quickly only at first, as shown in Peiser's sodium chlorid experiments, and later but slowly, to keep pace with the kidneys, as it were; a phenomenon which is a protection for the body against overwhelming amounts of bacteria and toxins. "If this equilibrium is disturbed by the introduction of saline solution into the peritoneum the animals die from a septic death from increased absorption, while the controls remain alive.

"In operative cases, it was also found occasionally that death occurred very quickly if the peritoneum was irrigated, so that the impression could not be avoided that this procedure caused direct damage." Rost says, "but whether the injury occurs in the sense of Peiser, or whether in spite of careful technic it is difficult to decide in a given case."*

The author confesses that at one time he advocated very strongly saline flushings during operation for acute peritonitis, and believed when one died that it was in spite of the treatment. After adopting Murphy's proposal, "quick in and quick out, prompt closure of leak, rapid approximation of the abdominal wound with a drain," the results were surprisingly better. Recently the automatic pump seems to be of value in removing septic fluids from the sac during operation. It is pleasing to have Peiser's experimental confirmation of our clinical findings in this work.

Prior to 1904 it was currently believed that it was necessary to remove all inflammatory exudates from the peritoneal cavity in acute peritonitis. Blake and many others, including the writer, held to this view, believing that this procedure removed the larger portion of the toxins in solution and considerable colonies of bacteria as well. By some, flushing of the abdominal cavity with hot normal saline was a routine in all cases coming to operation for acute perforative (spreading) peritonitis.

In a paper presented to the Southern Surgical and Gynecological Association at Birmingham in 1904,⁹ we made a plea for a common ground upon which we could all stand and the formation of some plan of treatment for this affection which could be accepted generally as the best method of treatment, giving the lowest ratio of mortality.

In the discussion Finney said, "My name has been mentioned by the author of the paper as advocating, some years ago, scrubbing of the intestinal coils in cases of this sort. A wider experience, however, has taught me that that method is entirely wrong."

In this discussion John B. Murphy startled his fellows by reporting

* Rost, *Pathological Physiology*, 245.

22 cases of acute general perforative peritonitis of spreading type with one death, and described the plan of treatment employed. Because of the fact that his report revolutionized the treatment of this affection and is almost universally employed at the present time, his remarks are given in detail:

"Included in these cases were perforations of the stomach, the duodenum, the intestine, typhoid perforation, ruptured periappendical abscess, etc., yet only one death.

"In the peritoneal cavity with pus infection, we have immediately after the dose is thrown in, so to speak, or immediately after perforation occurs, the element of depression with pain, nausea, or vomiting, which come from the immediate primary irritation of the peritoneum, which is sometimes accompanied by depression, rather than elevation of temperature, and even by a lowering rather than an elevation of the pulse. After an hour, or many, depending upon the virulence of the infective material, one begins to have abrasion of the endothelium of the peritoneum or blistering with rapid absorption of the infective products, which tend to a fatal toxic termination. Can we lessen that absorption? Yes, we can lessen the absorption of infective material by the blistered peritoneum on the same principle that we can lessen the absorption of toxins in other infected areas of the body. What have we done in the past? We have opened the abdomen in these cases, taken out and handled the intestines, and we have mopped off the layers or flakes of protective lymph. We have poured peroxide of hydrogen into the cavity; we have put in salt solution by the bucket; we have packed in yards of irritating gauze and sponges; we have done many other irrational and unscientific things. I have tried all of them and I have taken the opportunity of standing up and saying to bodies of medical men that I believed the great majority of such cases would die, no matter what I did for them. I want to take this occasion to apologize for having made that statement, and to say I was grossly in error. In the last three years I have had twenty-two consecutive cases of general suppurative perforative peritonitis, with one death.

"Why do these patients live now and why did they die before? I do not know, but they do live. I have no 'life-saving stitch'; I have nothing to offer you specially that will make them get well, except it be following a routine treatment which I do now, which is materially different from what I practised before. If the essayist finds that all these patients get well from washing out the abdomen, I would not recommend him to change his method in his work. I know that with the plan which I am following now these cases are getting well. My

fatal case was one of general suppurative peritonitis from periappendicular abscess. The patient contracted a double pneumonia on the sixth day, when the peritonitis had completely subsided, and died from it.

"In this discussion we have to consider the question, What can we do to lessen the immediate absorption? (a) Open the abdomen. (b) Shut off the source of supply of the infective material, whether that be a perforated stomach, a perforated duodenum, a perforated intestine, or appendix. (c) Do the least possible manipulation in the abdomen, the same as we do in a phlegmon of the thigh. (d) Put in a drainage tube. (e) Put the patient in a sitting (Fowler) position, to allow the infective material to escape into the pelvis and prevent it from being retained under pressure. The pressure element is a most important one, but it is not the sole element. (f) Empty bowel and stomach by sewage. (g) Give one and a half pints of normal salt solution every two hours. (h) If advanced toxic condition, give 20 c.c. Stern's anti-streptococcus serum at one dose.

"The gentleman who opened the discussion referred to another class of cases; namely, those in which there is streptococcus infection. I saw degrees of streptococcus infection formerly which I do not see now. I saw cases formerly in which it seems to me under the present treatment the patients would die. Those were what we might call cases of acute malignant streptococcus infection. They die from the rapid production and rapid absorption of the infective material. It is surprising to note the rapidity with which this infection enters the lymph circulation; this is not true of staphylococcus infection; the latter occurs with greater frequency in the peritoneal cavity. If I have treated the number of cases mentioned—twenty-three—in the last three years since I have adopted the plan referred to, with only one death, what does it show? That the number of malignant cases of streptococcus infection of the peritoneum at the present time is comparatively small, and if we can get results so favorable from such a plan of treatment, we ought to pursue that method and be satisfied without theorizing as to why we get such results.

"The details of treatment vary in cases of perforative peritonitis. If it be the stomach that is involved, an incision is made directly over it, and the perforation closed by two or three rows of linen sutures. If it be the duodenum, a similar plan of treatment is carried out. Immediately after closing the incision at the upper part of the abdomen down to the small drain placed there, a puncture is made through the rectus muscle, either the right or left, just above the symphysis pubes, and two large drainage tubes are passed into the most dependent por-

tion of the pelvis. No effort is made to flush or sponge out the abdomen. The patient is put in the exaggerated Fowler position. The treatment from this time is very important. In the first place they are either well on the way to recovery, or death will result within the first twenty-four hours after the operation. Then the question arises, Can you dilute the toxins or administer antibodies for the first forty-eight hours? In many cases I have observed advantage in the use of antistreptococcic serum. It does not seem to counteract the immediate toxic symptoms. In the second place, we should fill the large intestine with normal salt solution. I give patients as much as a pint and a half of saline per rectum every two hours. They get on an average of eighteen pints of salt solution in twenty-four hours. They absorb it all. They are not given anything by mouth, because the majority of them vomit after operation, and the stomach does not absorb water. We give strychnin, which is practically the only medicine administered. A large quantity of salt solution administered continuously by rectum dilutes the sepsis sufficiently to tide them over until their local immunity (tissue infiltration) is greatly increased. The infiltration of the tissues of the peritoneum which has been overpowered in the beginning undergoes such a change as to resist absorption (local immunity); this, together with the general leukocytosis, is so great as to overpower the sepsis and carry the patient over the dangerous first twenty-four hours, so that he can go on to recovery without interruption. Elimination equals absorption from that time, and the patient needs but little attention."

Following this discussion the writer has never found it necessary to employ irrigation of the abdominal cavity for this affection, with the most gratifying results. I wish to make this acknowledgment of one of Murphy's many valuable contributions to surgical knowledge.

This is the type of case in which drainage may be used with much benefit to the patient.

Drainage.—The trend of current surgical opinion is: "Lessen the number of cases in which drainage is employed." Many of these spreading, acute cases will recover if the intestinal leak is closed, the fluid removed from the abdomen by suction, or in some cases even when no effort is made to remove it. In other words, the protective ability of the peritoneum has not been fully appreciated.

John G. Clark demonstrated experimentally and clinically, first, that in chronic and even acute pelvic infections, drainage could be abandoned with safety. Subsequently it was shown that it was not necessary in some of the more acute conditions where further contamination was checked. Drainage is indicated in cases where the endothelium is much

injured and where considerable dead tissue must be taken care of, rather than for the cases with large accumulations of fluid within the peritoneum.

Drainage has been employed to remove material from the abdominal cavity, which could not be taken care of by the protective forces of the peritoneum, or rather the removal of the material which the attendant believed would not be taken care of in such way. Increasing experience always diminishes the number of drains employed by a surgeon. It may be said that the amount of drainage employed is inversely the measure of the technic of the surgeon.

The reasons why drainage was employed by the early aseptic surgeons was the fear upon their part of the ability of the patient to take care of the microbic contamination and necrotic tissue. In the early days of abdominal surgery, practically all cases of celiotomy were drained, either by means of a glass or rubber tube. The fluid was removed by a suction syringe, carried down to the bottom of the pelvis every one-half to one hour for two or three days, at which time the tube was removed.

With increasing knowledge there has been a steady lessening of the employment of drainage. The insertion of drain is an acknowledgment of a weakness in our technic. One says, "I do not feel sure of this abdomen, so will leave a vent."

Originally it was believed that all the poison could escape from the abdominal cavity through the drain. Just as the possibility of washing the cavity free from infection was found to be a fallacy, so is it a fallacy to think the entire cavity can be freed by drainage.

To understand fully the question the physiology of absorption must be understood. The process by which fluids in the cavity are taken into the blood stream is not always simple of explanation. A portion of this process is one of osmosis, in which the peritoneal structure acts as a dialyzing membrane. In such a condition the more concentrated solution receives fluid from the less concentrated until a balance is obtained, while at the same time the inorganic constituents in the more concentrated solution pass into the one of less density. When the concentration of the blood is high, fluid promptly enters to diminish its density until it becomes isotonic with the abdominal fluid. At the same time there is a relative diminution in the saline ingredients of the blood and a proportionate increase in that of the peritoneal cavity. It must be remembered that albuminous fluids do not pass readily through a dialyzing membrane, hence the accumulation of large quantities of fluid within the abdomen containing a high percentage of albumin.

Pus is not readily removed by the absorbent vessels, either lymph or blood-vessels. It can only be taken up rapidly when just a few cells and a large amount of fluid are present, as in the early stages of an acute peritonitis, or very slowly when it consists of a highly albuminous fluid with many dead cells.

Drainage is largely a mechanical process based on the propensity on the part of a fluid to reach its own level—for this reason drains have usually been placed in the lower portion of a wound—in the abdomen placed so the bottom of the pelvis can be drained. The assumption is that all intestinal fluids gravitate to this point, while the larger number of absorbent vessels is found in the upper portion of the abdomen.

In addition to gravity something is needed to carry the fluid up hill from the bottom of the pelvis to the abdominal wall. To accomplish this two factors are utilized, one the intra-abdominal pressure above and a siphonage effect upon the fluid within the pelvis. In order that the drainage of fluid from the pelvis may be encouraged it has been found that by keeping moist gauze about the outer end of a wick drain a marked increase in the flow may result especially when proctoclysis is employed. It has also been found that the application of moist packs to the wound will keep the flow up for twenty-four or forty-eight hours. If, however, a drain is placed in the wound and dry gauze placed over it, drainage is practically nil. This seems to point to the advantage of siphonage. These findings lead to the conclusion that the practical effect of drainage is small. Mainly it provides a vent.

What takes place when a drain is placed? There is immediately an outpouring of serum and of lymph from the surrounding tissues and agglutination of the peritoneal coverings of the intestine and of the mesentery, to each other in such a way as to form a tube or canal about the drain, separating it from the remainder of the peritoneal cavity. As soon as this is accomplished the efficacy of a drain is lost. A way is provided, however, by means of which purulent accumulations may find egress.

By observations such as the above, surgeons have come more and more to the conclusion that drains are often unnecessary. An exception to this is the presence of a leaking intestine, gall-bladder or urinary bladder in which closure cannot be accomplished. Escape to the surface is aided by a drain until canalization takes place.

Drains will also be needed in cases where pus cavities persist and where large necrotic masses remain.

There are a number of cases in which a large appendicular abscess with localized peritonitis is present in which drainage is imperative.

There is such a large dead space to be taken care of that the natural resistance cannot cope with it without aid. In such cases the application of hot packs is essential in order to restore the circulation to normal and to promote the escape of pus.

The drain in such a case should remain somewhat longer than in the cases of free fluid in the abdomen because the pus flows out less freely, and if the drain is removed too early pocketing may occur with several small abscesses and finally spread the infection. When the tube is removed no effort should be made to wash out the sinus or to replace the drain. The abscess can only heal by the collapse of its walls, which occurs rather promptly from intra-abdominal pressure. In these cases the gentlest possible pressure near the drainage tract will bring the contents of the abscess to the surface. In a short time the discharge ceases and the temperature remains normal. When these conditions present and the drainage opening shows healthy pink granulations and no pus the edges may be brought together by adhesive (zinc oxid) plaster.

During the entire healing, gaping of the wound and the tendency to hernia later may be prevented by keeping the abdomen well strapped up. Very rarely, if proper suture methods are employed in such cases, the wound edges may separate and the intestines escape. Under such circumstances the intestines should be protected, gently cleansed with normal salt solution, and after releasing any adhesions to the wound edges replaced within the abdomen. This accident has occurred so infrequently in my service that I never use stay sutures for purposes of relaxation and additional support. Where a strong reliable brand of prepared catgut is used the wounds will hold. We have found a No. 2 chromic will withstand the strain in these suppurative cases satisfactorily until the wound is completely healed. This description covers rudimentary principles and our method, though somewhat different from those in vogue, is most satisfactory. It meets the indication and has not the objection of stitch abscesses from nonabsorbable sutures. Moreover, the patient has none of the dread of pain from the removal of the stay sutures.

We consider that any experienced surgeon knows when a granulating wound is ready for secondary suture by the appearance to the eye. There is no objection, however, to taking smears from day to day and counting the number of bacteria to a field before closing secondarily. When the bacteria are as low as five to ten to a field it is usually safe to close.

In the treatment of all abdominal wounds the most generally useful

germicidal agent is 60 per cent alcohol. In order to dry the skin and inhibit bacterial growth iodine, $3\frac{1}{2}$ per cent, is useful prior to operation, but alcohol is subsequently most valuable. To remove the adhering zinc oxid plaster fragments, benzine or gasoline is a good cleansing agent.

When the discharges, either pus or feces, are irritating to the skin nothing equals oxid of zinc ointment as a protective. Other than this all powders, pastes, etc., should be discarded.

In order to prevent hernia subsequently when the wound is extensive it may be of advantage to provide for a well-fitting abdominal support which should be worn for some months. In most cases where the healing is prompt there is no necessity for such support.

It is a far cry from the silk pedicle ligatures hanging out of the abdominal wound employed by Ephraim McDowell, Nathan P. Smith, and by Clay of England, the glass drain of Koeberle, Saenger's use of a wick inside a glass drain, Fritch's simple gauze drain, von Mikulicz' gauze bag, and many others, to the closure of the abdomen with absence of drainage in a very large proportion of cases at the present day.

John G. Clark, in 1897, reported 1700 cases of abdominal section performed in Johns Hopkins Hospital from its opening in 1889 to October 1, 1896, and concluded that not only is drainage useless in the great majority of cases in which it had been previously used, but that it is frequently productive of harm.

In the first Gynecologic Report of Johns Hopkins Hospital, H. Robb gave five indications for drainage, and because of these indications 73 per cent of all sections were drained.

As a result of the bacteriologic investigations of Robb, and a more extended clinical experience with the tube in the second hundred cases, drainage was employed in 57 while in the third hundred 48 were drained.

About this time Kelly began to have cover-glass specimens made at operation, and when bacteria were found drainage was employed. Following this in the twelfth, thirteenth, and fourteenth hundred series the percentage of drained cases was larger.

Later the great frequency of infection of the drains as demonstrated by bacteriological studies by G. B. Miller and Clark and the variety of living pyogenic organisms in the diseased structures as shown by Miller caused almost a complete abandonment of drainage as a measure of removing infectious matters from the peritoneal cavity.

Clark's experience is confirmed by the larger number of workers in this field during this period. He proved that drainage could very largely be dispensed with when abdominal sections were performed with due

care. To accomplish such result he claimed the following conditions must be met :

1. Thoroughly disinfect the hands.
2. Control hemorrhage.
3. Avoid bruising or otherwise injuring the tissues.
4. Isolate the general peritoneal cavity during operation.
5. Preserve the peritoneum.
6. Conserve the bodily heat.
7. Avoid rupture of intraperitoneal abscesses.
8. Irrigate the peritoneal cavity.
9. Promote absorption by saline infusions into the peritoneal cavity, followed by postural drainage.
10. Use submammary saline infusions.

All of Clark's postulates with the exception of two (8 and 9) may be accepted at the present time. Since his paper was written, sterility can be practically guaranteed by the proper preparation of the hands and the use of rubber gloves.

The control of hemorrhage must be absolute and not left to chance, since the blood clots remaining after considerable oozing are active causes of suppuration.

A careful avoidance of insult to tissue is of extreme importance, thus preventing the necessity for the disposal of large amounts of dead tissue within the peritoneum. Denuded surfaces should be repaired and covered with healthy peritoneum when the conditions will permit.

We have found that it is quite possible to protect the peritoneal cavity from pus by surrounding the abscess with gauze and taking up the excess fluids either upon gauze sponges or by means of an automatic pump, and even where drainage is necessary, wounds heal kindly throughout except at the drainage tract.

Irrigation of the peritoneal cavity with fluids during operation has been almost universally discarded. The same is true of the instillation of saline solutions into the cavity before closing the wound, but Fowler's position is very extensively used in connection with the Murphy drip proctoclysis methods with a view to delaying absorption of toxins from the abdomen and filling up the vessels with fluid after operation.

Submammary saline infusions advised by Clark still remain in general use to-day.

III Effects of Drainage.—1. Postoperative obstruction often occurs from the formation of adhesions about the drainage tract and results in the necessity of secondary operation to relieve obstruction.

2. Where drainage is extensively used fecal fistula is seen quite fre-

quently as a result of the pressure from a glass or rubber tube or from the tissues becoming enmeshed in the gauze, which may cause a rent in the vessels when it is removed.

3. The presence of a drainage tube pressing upon the bladder often causes vesical irritation.

4. Postoperative hernia is not the least frequent nor the least important sequel to its use.

Indications for Drainage.—1. The presence of free purulent material in considerable quantity in the peritoneal sac.

2. The presence of an abscess sac with damage to the peritoneal surface.

Under the first group the weight of professional opinion favors the employment of drainage. In cases where the fluid is but little tinged, yellowish, or almost clear, we have found drainage to be unnecessary. It is also true that when in appendicitis the organ is discolored, distended, and even with evident free fluid in the cavity, drainage may be discarded.

It is not necessary to drain cases of ruptured duodenal or gastric ulcer. In most cases of typhoid perforation drainage may be left off if the rent is closed. Where there is a large amount of material to be taken care of, some operators drain.

Perforating gunshot wounds of the abdomen are very seldom drained and then only to meet some distinct indication. Very rarely is drainage indicated in intussusception, volvulus, or mechanical ileus. In operations for acute puerperal peritonitis of severe type with free sanguinolent fluid in the abdomen, drainage is indicated. The same is true if operation is performed in the very early stages of an acute gonococcic peritonitis.

Contraindications for Drainage.—There is no indication for the use of drainage:

1. In all clean cases: in all cases where the intra-abdominal fluid is clear or shows but little cloud or yellowish tinge.

2. Where there is no break in the peritoneal surface.

3. In the absence of a large amount of dead tissue.

4. In all acute conditions where the soiling of the peritoneum is limited.

5. When the toilet of the peritoneum is satisfactory to the operator.

6. When the nature of the infection is not a virulent one as proved by the clinical picture, by the anatomical situation, by a well developed leukocytosis and by the laboratory findings.

Robert Morris was among the first to recognize the protective power

of the peritoneum. With increasing experience one recognizes more fully the ability the peritoneum has to take care of foreign material.

The earnest and skilled surgeon soon can estimate the cases in which drainage is not necessary. When in doubt do not drain, because the indications are so positive when its employment is *necessary* that no one can be in doubt when to use it.

Materials Employed in Drainage.—The use of glass, metal tubes, and hard rubber drains has been abandoned. In the small number of cases needing drainage, the soft rubber cigarette drain carrying a wick of gauze within it is most satisfactory. A firmer rubber drain is occasionally employed. Some surgeons split it throughout and place a gauze wick in it. In the use of these drains the end of the gauze should be flush with the pelvic end of the tube.

In rare instances gauze alone is used, but it has the disadvantage of forming adhesions to the peritoneal surface of the bowel, and in removal may tear the wall and a fistula result.

After drains are placed in the manner described they should remain in situ a varying period of time, from twenty-four hours to four or five days. When only serum escapes from the drainage tract after twenty-four hours the wick may safely come away. Where a large amount of necrotic suppurative tissue is to come away the drain may remain seventy-two to ninety-six hours, or even longer. If the drain is made of gauze alone, and because it becomes enmeshed in the tissues it is usually left longer. About the fourth or fifth day a certain amount of serum is poured out around it separating it somewhat from its attachments, and it comes away much more easily than it would earlier.

As stated before when the silk ligatures were used to tie pedicles and sometimes remained a long time before they could be loosened and brought to the surface by the action of the tissues alone, it was found that the use of peroxid of hydrogen would hasten their expulsion. We have not found it necessary to use this procedure for many years past.

Postoperative Care.—Subsequent to the immediate measures employed for shock the two most valuable remedies in its management are gastric lavage and the rectal instillation of normal saline solutions. I fully realize that each of us has to have his particular fad, and if I am forced to ride one this should be it.

Postoperative nausea results from efforts at elimination of the anesthetic through the stomach. In addition, this organ takes care of any back flow from the intestine by emesis. All cases of stoppage of the fecal flow, whether due to mechanical or adynamic ileus, produce very poisonous toxins in the gut. For years we have recognized the fact that

toxic products within the intestine in peritonitis were equally if not more depressant to the cardiac apparatus and to the nervous mechanism than those within the peritoneal sac. It has been the custom in our clinic to perform gastric lavage in all serious cases of peritoneal inflammation. In those operations performed for relief of ileus and for general peritonitis with but few exceptions gastric lavage should be used prior to operation. It prevents gaseous interference with respiration from pressure; it prevents the patient from drowning in his own vomitus during anesthesia. It also, when used after operation, prevents distention, makes the patient comfortable, and checks nausea and vomiting. When it fails in those three efforts after repetition there is very strong presumptive evidence that a mechanical ileus is present. Rarely does adynamic ileus fail to show improvement under its use.

The so-called acute dilation of the stomach which in my opinion is in most cases due to infection and to a paresis from peritonitis, may in nearly every case be relieved by repeated washings with hot water to which a small amount of sodium bicarbonate has been added.

Those cases which show a rapid pulse, some prostration and cold sweat after operation will be much benefited by the Murphy drip as previously described. One to one and one half pints given at intervals of four to six hours will be most efficacious. The subcutaneous instillation is employed when proctoclysis is indicated, but is retained poorly.

Certain cases do so well that the use of saline solutions in this way is not necessary. When after the first rally of the patient's vital forces, the pulse becomes faster, the temperature goes higher, this method should be again employed.

The profession, too, seems slow to arrive at the conclusion which we reached a number of years ago concerning purgation after operation for intra-abdominal lesions. It is unnecessary to worry about a bowel movement in the great majority of cases since this occurs at the proper time as a general rule. In cases which are otherwise doing well and where it is felt that a purge is indicated a dose of *Ol. Ricini* is given on the third or fourth day. This is done not as a routine but because in the opinion of the attendant it is indicated.

If a drain has been placed in a wound this may be removed on the third morning without disturbing the main dressing unless it is soiled, or has considerable odor. The drain may remain in situ much longer when much dead tissue or an abscess wall is to be cared for. It should come out whenever it has ceased to fulfill its function or when it becomes irritating.

Clean abdominal wounds should not require a change of dressing for

a week or ten days, in fact until healing is complete. Hemorrhage calls for immediate dressing and so does pain in the wound. The latter symptom when sharp and continuous means either hemorrhage or infection. A clean dry wound is not usually painful.

The patient's convalescence may be materially hastened by the early assumption of the sitting posture. The patient reasons, and properly, that if the case is serious the surgeon would keep him flat and quiet. How patients were punished in the early days of abdominal surgery! They were purged as much as they could stand, depleted, dehydrated, and starved prior to elective operations. After the operation they were placed flat and kept immobile, were prevented from getting any water by mouth or even wetting their lips for twenty-four, thirty-six, or even seventy-two hours. The nausea continued until the anesthetic was eliminated and the toxins in the stomach and bowel were disposed of by emesis. Their backs ached from the irksomeness of their fixed position. Each one felt as if he would die, cared but little if he did, or feared that he would not. The dread of the time for the removal of the stitches and in addition the daily dressing and the hourly suction from a glass drain made for anything but a comfortable convalescence.

Contrast that picture with the usual cheerful happy convalescence of the present day when the patient is sitting up in bed taking water from the first with very little discomfort of any kind, and we are compelled to prefer present to former practice.

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CHAPTER V

PERFORATIVE PERITONITIS

TYPHOID ULCER

Perforation from typhoid fever is very much less frequent than it was some years ago, largely because of the control of this disease, resulting from persistent efforts of the various health boards and sanitarians. From a very frequent affection typhoid fever has become one of the rarer diseases, particularly in those communities in which the water supply is properly obtained and examined at frequent intervals. The usual percentage of perforation of typhoid ulcer to the number of cases of the disease will probably persist. This complication adds very greatly to the gravity, having a high mortality, both from the peritonitis *per se* and because of the depletion of the sufferer before perforation occurs. Fortunately, impending perforation during the course of typhoid fever often announces its probable occurrence to the attendant by a marked and increasing meteorism, exaggeration of the local tenderness, in some instances amounting to localized pain near McBurney's point. These symptoms developing in a patient who has been doing comparatively well previously, should always put the attendant on guard against impending perforation. Unfortunately there is little or no increase in the leukocyte count as compared with the normal, but if the leukocyte count has been 5500 or 6000, and becomes increased to 6500 or 7000, it becomes quite suggestive. Add to this muscular rigidity in a previously flaccid abdominal wall, and one should not be surprised when complaint is made of sudden intra-abdominal pain. This symptom, with a sudden fall of temperature, in some instances three or four degrees, with the evidences of shock, cold, clammy sweat, and marked prostration makes the diagnosis clear. It must be recalled, however, that intestinal hemorrhage produces collapse, prostration, and lowered blood pressure closely simulating perforation, but it is rarely accompanied by pain, and is soon evidenced in the stool. The history and classical symptoms of typhoid fever will help to make the diagnosis from hemorrhage, from appendicitis or other sudden intra-abdominal calamity. The leukocyte count in appendicitis will be much higher and has a sudden onset, in a previously healthy patient.

Other intra-abdominal calamities can usually be differentiated from typhoid perforation by the clinical history and local symptoms, together with the acuteness of the process.

Rose spots, when present, are strongly indicative of typhoid fever. The Widal reaction is practically conclusive of its presence. However, typhoid fever may exist and carry an acute perforative appendicitis as a complication. It is well to bear this fact in mind.

The management of perforating typhoid ulcer demands good judgment. These patients are exceedingly poor surgical risks, since typhoid fever itself carries a high mortality without this serious complication. A leak from this highly inflamed intestine into the peritoneal cavity makes a fatality almost a certainty, unless something can be done in a mechanical way either to prevent the leak, or to close it promptly and to protect, as far as possible, the clean peritoneum from further contamination. This will necessitate immediate operation just as soon as the diagnosis of perforation is made or in some instances when the symptoms indicate that it will probably occur. Of course, no one would advise intervention without the most cogent reasons.

Undoubtedly, the safest method of handling a case of this kind is under seminarcosis from opium with local anesthesia. An opening in the midline or right semilunar should be made, the ileum near its junction with the colon picked up, the leaking ulcer located, sutured carefully, several wick drains inserted and the abdomen promptly closed. The procedure should only take a few minutes. The enlarged lymphatic glands in the mesentery will aid in locating the loop. Perhaps in some cases bringing the leaking loop of intestine into the wound after placing a rubber tube into the gut at the site of the perforation might prove satisfactory, but this necessitates a second operation for the relief of the enterostomy. The advantage of the enterostomy tube would be relief of meteorism, drainage of the infected intestine through the tube, and perhaps the advantage of placing therapeutic agents in direct contact with the ulcerated bowel. The subsequent treatment would be that of acute peritonitis, with such medicinal measures as are indicated in typhoid fever.

GASTRIC OR DUODENAL ULCER

A consideration of the subject of peritonitis would not be complete without a study of gastric and duodenal ulcer in their relation to this affection.

Either of these lesions may be a causative factor in the establishment of peritonitis, particularly of the acute perforative type. Unless due con-

sideration is given to these conditions in the determination of the causative lesion one may be led widely astray, both as to accuracy of diagnosis and to methods of treatment.

Both gastric and duodenal ulcers are essentially chronic in character as a rule. Occasionally, however, they may run a brief and acute course. Such lesions may exist for long periods of time and produce no changes in the peritoneal cavity. In the meantime the patients are suffering very greatly from dyspeptic symptoms.

It is really remarkable how resistant to ulcer the peritoneal portion of the gastric wall is. The author recalls a case in which the diagnosis of gastric ulcer was easily and accurately established, but operation was not accepted by the patient. Temporary improvement from rest and diet with some of the usual medication occurred. Then followed symptoms for two years, when he was again seen and operation was considered, but again refused. A short time later his ulcer ruptured and he was again seen. At this time operation was out of the question, the patient being moribund, and death occurred in a few hours. Necropsy showed a very large circular punched-out ulcer involving mucosa and muscularis 7 cm. in diameter. In the center of the crater of the ulcer, the base of which was formed of the serosa only, there was a rupture of the serosa about one inch in extent. It was very evident that this ulcer had for a long time rested on the peritoneum alone. This clearly shows the strength of the peritoneum and its resistance to this type of pathology. It also points particularly to the long period of warning which these patients have before the dire disaster seizes them. A very large number of patients dying from so-called acute indigestion soon after a large meal undoubtedly die from rupture of a gastric or duodenal ulcer.

In view of present knowledge and because of the prodromal symptoms antedating perforation of these ulcers, this type of peritonitis becomes a preventable affection.

The methods of detection of a gastric or a duodenal ulcer are so accurate that few are overlooked by competent and painstaking observers.

Fortunately peritonitis resulting from leaks of this character differs materially from that resulting from acute appendicitis. Two factors are active in rendering perforations here less virulent than in lower portions of the alimentary tract, particularly those from typhoid fever and from appendicitis. One of these factors is the inability of the individual suffering from ulcer to take all kinds or quantities of food. The other lies in the fact that for the most part the contents of the stomach and duodenum are sterile.

Immediately after a meal bacteria are found within the stomach, but

in a short time they practically all disappear. When, therefore, rupture occurs as is usual within a few hours after a meal the exudate is in many instances sterile. This rule is not absolute and some cases show bacterial flora. Dugeon¹ found streptococci present in 12 of 23 cases. "The organism was of low virulence or non-pathogenic to animals." He found *Staphylococcus albus* in 10. The exudate was sterile in 5. He notes, as have others, that when colon bacillus is present, the infection is more severe.

Some ulcers form adhesions about their base, and when perforation occurs the localized peritonitis previously existing protects the peritoneal cavity against a spreading contamination and a localized abscess forms.

The time at which rupture occurs is highly important as regards toxicity of the exudate. Ruptures occurring late during the time of stomachic digestion are often more or less innocuous because of the small number and low virulence of bacteria present. This is one reason why very few operators drain the peritoneum when operating for ruptured duodenal and gastric ulcers. The peritoneum is able to take care of this type of infection if the primary leak is promptly controlled. When rupture occurs promptly after the ingestion of a large meal with plenty of fluids, and where the amount of extravasation is great, the peritoneum is less able to deal with the contamination.

Another reason why the peritonitis following a leak from gastric ulcer is not drained is because of the location of the rupture. In most cases this occurs on the posterior wall and the exudate is more or less limited to the lesser peritoneal cavity. Unless this is remembered, at operation this form of lesion may be overlooked and the case be considered as of unknown origin. The lesser curvature or the anterior wall may be the site of the perforation in a smaller number of instances.

Cases with the rupture in the latter locations promptly excite a general peritonitis from the wide extravasation of a considerable quantity of fluid.

Ulcers of the duodenum are probably better taken care of than those of the anterior wall of the stomach. The relations of the duodenum and its fixed position make isolation of its exudate rather easy. Moreover, after stomach digestion, the contents are often innocuous in this region. Obstruction below the point of rupture tends to exaggerate the damage resulting because of the greater amount of food retention.

Leaks from rupture of the duodenum tend to follow the right colon to the right lower quadrant. This causes the symptoms to simulate appendicitis. Those occurring behind the stomach may escape through the foramen of Winslow and simulate gall-bladder inflammation. Those

occurring on the anterior wall of the stomach tend to collect in the left flank. Agglutination to the anterior parietal wall may result in a localized abscess in such a case. Localized abscess may form in right lower or left lower quadrant, or in either flank. Sometimes pus accumulations are found between the liver and the diaphragm.

Symptoms.—The clinical picture of a perforative peritonitis from gastric or duodenal ulcer is rather typical. In the very largest number of cases there is a history of dietetic distress, sour stomach, eructations, belching, hunger pain before, and actual pain and discomfort after eating. This pain comes on at varying intervals after the ingestion of food, depending in part on the location of the ulcer. If at or near the cardia it occurs soon after eating. Water alone may excite it. If near the pylorus it appears later. If due to duodenal ulcer it occurs after two or three hours.

Localized pain over limited area, transmitted through to back is quite characteristic. Tenderness in same locality is usually present. Vomiting is of frequent occurrence and often gives relief. Vomiting of blood may have occurred. These patients take large quantities of alkalies for the sour stomach. Certain types of food disagree.

All cases do not show these symptoms. Duodenal rupture may occur suddenly with but little previous discomfort.

Blood, either as tarry stools or in microscopic amount, may have been present in the feces. Blood may be found in the stomach or duodenal washings. It may be visible or detected by the microscopic or chemical examination (occult blood).

These patients with an extended history have usually lost flesh. The appetite is vigorous but capricious.

When perforation is impending the patient is anxious, has a sense of foreboding. Something is wrong but he may not locate it. The onset of peritonitis is evidenced by sudden, sharp, severe pain, continuous in type and localized in the epigastrium. It is not cramping in type. Later it becomes general and general abdominal tenderness persists. The shock is usually marked. It is much exaggerated if hemorrhage occurs at the site of rupture, after or prior to its occurrence. Vomiting is often absent soon after rupture occurs.

The abdominal wall is scaphoid from contraction of the muscles, due to reflex muscular contraction from the pain and to protect against its exaggeration by bodily movements or from the respiratory excursions.

The diagnosis of such rupture as causative of the advancing symptoms of peritonitis is to be made from ulcer with no tendency to perforation. Usually there has been considerable pain and the quality of this pain is

recognized. The pain of rupture is not relieved by vomiting, the ingestion of food or soda or any of the usual remedies previously successful in its treatment. Marked boardlike rigidity is characteristic of perforation.

Errors have been made in some instances in which the gastric crisis of tabes has been mistaken for the more serious rupture of a gastric ulcer. The symptoms of gastric crises are never as severe as from a leaking gastric ulcer. The attacks of pain in tabes occur most often at night. They are not accompanied by shock. Girdle pains are a usual accompaniment of luetic crises. Diminution or loss of patellar reflex is evident and the tabetic gait is observed. Romberg's sign is present. Some evidence of incoördination is observed. None of these nervous phenomena is present in ulcer. It is only important to call the attention to the possibility of this unfortunate error and the incidental embarrassment it may cause.

The proper care of gastric or duodenal ulcer is important as a measure for the prevention of peritonitis.

This of necessity includes the medical care since it must be admitted that some peptic ulcers may be cured in this way. We cannot concede that the observation of Frick³ that the large majority of peptic ulcers which are free from serious complications will heal under medical treatment is correct. The percentage of recoveries under such treatment is small and recurrence frequent.

Dietetic and medical measures should be employed for a reasonable period and surgical methods promptly instituted when such treatment fails of prompt and permanent relief.

According to reports of Rosenow, confirmed by others, bacteria may be recovered in pure strain from peptic ulcers. When such a culture of bacteria (streptococci) is injected into the ear of a rabbit a peptic ulcer of the stomach can be produced. This seems to prove the contention of Rosenow that the cause of peptic ulcer is derived from infarction resulting from focal infection. This also points to the importance of the determination of the original focus and subsequent foci of infection and their removal as a part of both the medical and the surgical treatment of this disease.

Frick contends that no alkalies need be given in the medical treatment of gastric ulcer. Nevertheless, he advises the use of bismuth subnitrate in large doses, which is probably of much value. Carefully regulated diet is also important both before and after surgical treatment is undertaken.

Operative measures are indicated in peptic ulcer for persistent hemor-

rhage, for stenosis and retention, and for the prevention of rupture and peritonitis in cases which are more or less intractable.

The management of such an ulcer at operation requires judgment as well as technical skill. Ulcers in which impending perforation is probable in operations made for cure of the condition should be excised when possible. The actual operative steps in this procedure vary greatly, depending upon the extent, the character and the location of the ulcer.

Small ulcers readily accessible may be excised and the gastric or duodenal wound closed by suture. This may be easily accomplished when they are situated upon the anterior wall of these organs. Small ulcers upon the posterior wall of the stomach may be excised through a trans-gastric incision.

Inaccessible ulcers may in some instances be rendered accessible by mobilizing the duodenum by an incision along its right superior border. In this step care should be observed to avoid the large vessels. When impossible to avoid them they should be doubly clamped and ligated.

Ulcers of considerable size involving the pyloric portion of the stomach may be safely resected after this plan. Subsequent to the excision a gastroduodenostomy may be possible and satisfactory. In a considerable proportion of cases, however, dependence must be placed upon closure of the gastric or duodenal wounds and completion of the procedure by a posterior gastro-enterostomy.

More extensive procedures may be accomplished prior to rupture of the ulcer than are possible after this accident. The gravity of the patient's condition following rupture of a gastric or duodenal ulcer may be so great that only the smallest possible amount of surgical interference is permissible.

It has been found that simple closure of the leaking ulcer will in a large number of cases result not only in temporary but often in permanent relief. Just why such result obtains is not easy of explanation. Certain factors apparently enter into such result. Among these may be mentioned the circulatory changes about the ulcer following the accident and the efforts at surgical repair. Certainly a temporary increase in the blood supply to the part results in bringing increased reparative changes locally. Undoubtedly the restriction of the diet and the rest in bed incident to the operation are important aids to healing, while therapeutic agents administered during the period of convalescence are also a factor in recovery. Cases which fail to heal after simple suture and in which gastro-enterostomy is indicated and primarily possible, may be treated by this measure after closure of the leak.

Posterior gastro-enterostomy by the no-loop method of Mayo is ac-

cepted as the method of choice. In its performance certain precautions are observed. The field should be protected from leaks as in all gastrointestinal surgery. The blood supply to the wound edges is to be preserved. The sutures of coaptation should control hemorrhage and at the same time should not constrict the tissues to the impairment of their nourishment. Most surgeons now rely upon catgut suture material. Silk is thought to favor the development of jejunal ulcers which occasionally occur as sequels of this procedure. Proper placing of the opening in the stomach and jejunum to a large degree avoids this occurrence.

Judd and Rankin⁵ have recently advised the use of resection of duodenal ulcers and direct suture without gastro-enterostomy to avoid the formation of secondary jejunal ulcers. They say: "In the light of the present knowledge of the cause of gastric and duodenal ulcers, it is logical to remove the offending lesion together with the foci of infection, rather than to trust to a side-tracking procedure which leaves a pathological entity in which healing is a moot question and in which exacerbation gives rise to a chain of symptoms similar to those for which the patient first sought relief."

They also state that in the cases treated by this method there has never been an occasion to supplement the operation by gastro-enterostomy because of pyloric stenosis.

For the purpose of accomplishing the same results Finney² has presented a new method of end-to-end suture in performing gastroduodenostomy, and states that Hans Haberer⁴ used an almost identical procedure. In performing this operation after resection of the ulcer, the freely mobilized duodenum is separated from its loose areolar attachments and the open end of the stomach is sutured into the side of the duodenum. In the curative treatment of this disease prior to rupture, excision is the method of choice, gastro-enterostomy that of necessity. Some gastric ulcers of the lesser curvature or posterior surface attach themselves at their base to the liver or to the pancreas. This materially adds to the difficulties of dealing with the ulcer.

W. J. Mayo⁶ employs the approach to the posterior surface of the stomach from above. He divides the gastrohepatic omentum, ties the gastric artery if necessary, and separates the adhesions on the lesser curvature to secure sufficient operating space.

After all adhesions are cleared away with the finger around the adherent ulcer, he hooks the stomach and pancreas in such manner that they can be drawn into the wound and made accessible. The stomach is held up by the finger or gauze tape and the ulcer shaved off from the pancreas deep enough to include all the base. When all the base cannot

thus be exposed in large ulcers, the pancreatic defect is carefully seared with the cautery. The stomach is carefully sutured.

The clinical results of simple gastro-enterostomy in some cases of gastric ulcer with such widespread infiltration that the suspicion of malignancy is strong, are at times surprisingly good. It must be admitted that healing of the nonmalignant ulcer or ulcers following gastro-enterostomy occurs in a considerable proportion of cases. The exact reason for this result is not entirely clear. However, in view of the fact that ulcers usually heal when pyloric resection is done without a gastro-enterostomy it would appear that doing away with pylorospasm is quite important. This is certainly accomplished when the pylorus is closed and a gastro-enterostomy is employed. It appears reasonable to conclude that gastro-enterostomy without closure of the pylorus favors repair of the ulcer by allowing prompt evacuation of the stomach contents and the cessation of pyloric spasm. It also prevents hyperacidity by drainage of the gastric contents. The latter condition must play an important rôle in the persistence of an ulcer. Notwithstanding the fact that normally the acid gastric juice does not damage the stomach wall it is demonstrated that it does effect the healing of an ulcer. This is particularly true when the percentage of acid is extremely high. The high acidity causes active pylorospasm and the contractions also tend to keep an ulcer open. The ulcer in turn excites hyperacidity. Gastro-enterostomy by changing the character of the gastric contents breaks this vicious circle.

The treatment of perforation from gastric or duodenal ulcer calls for prompt abdominal section. The mortality increases with each hour of delay in closing the leak. The section is made in the median line in the epigastrium or transrectus, about half an inch to the right of the midline. After cauterization or without it, the site of the perforation is sutured carefully to guard against further escape of fluid. Usually a gastro-enterostomy is not necessary. After a careful peritoneal toilet the abdomen is closed without drainage. In exceptional cases with considerable soiling as after a meal, a drain may be employed.

RUPTURE OF URINARY BLADDER

The urinary bladder is not so closely related to the development of peritonitis as some of the other organs. Occasionally, however, it is an active causative factor. This is particularly true of rupture of the bladder from traumatism, as blows upon the abdomen, and from stab or gunshot wounds. Fortunately such conditions occur infrequently.

It is really remarkable how much distention a bladder will withstand before rupture will take place. Even in case of close stricture rupture of the urethra takes place more often than the bladder itself.

Rupture of the urethra with urinary extravasation into the cellular planes may produce a severe phlegmon which occasionally results in a peritonitis. The chief importance as exciting peritonitis must be placed upon traumatism of the bladder.

The normal urine is perhaps less irritating to the peritoneum than extravasated intestinal contents, but it is unsafe to permit it to remain long in contact with the peritoneum.

Whenever suspicion of injury of the bladder is present this possibility should be carefully investigated. The failure to pass urine is a more important symptom of injury to this organ than even the presence of bloody urine. The catheter should be used to determine the functional capacity of the bladder. The absence of urine and the presence of some blood in the bladder are strong evidences of injury. Cases are on record where a rent in the bladder wall has been temporarily plugged by intestine or omentum agglutinated to the wound, occluding it and retaining urine within the vesicle. Cystoscopy or radiography may be called into use to clear up a doubtful diagnosis.

The treatment consists of immediate abdominal section with closure of the rent.

Occasionally the bladder is injured during operative manipulations in abdominal section. It must be remembered also that the bladder may be present in a hernia, particularly of the sliding type. Under these circumstances the greatest care must be exercised to avoid injury. The presence of large venous trunks and a very thick fold of tissue in a hernial sac should call for orientation before incising it.

Wounds of this character occurring accidentally during operations require careful suturing with catgut. Fortunately they rarely cause any inconvenience, healing taking place per primum. Subsequently a catheter may remain in situ for a few days, but this may not be necessary.

THE GALL-BLADDER AND PERITONITIS

Disease of the gall-bladder is important in connection with a study of the peritoneum, since infection may pass through its walls and excite a peritonitis, and because it may rupture as the result of traumatism from overdistention, necrosis or ulceration through its walls and excite such an inflammation. It is also productive of adhesions in many instances which may prove troublesome or even dangerous, causing stasis or

mechanical ileus. Infections in the gall-bladder often precede and are causative of severe pancreatic lesions. In prolonged cases of gall-stone disease the possibility of the development of malignant disease is by no means small and extension to the peritoneal structures may occur. The complete investigation of gall-bladder diseases is beyond the scope of this study, but the salient features should be considered with a view to the prevention of peritoneal affections arising from this source.

Even the simpler forms of gall-bladder inflammation may excite a localized plastic peritonitis with adhesions to the surrounding structures. Such condition may occur promptly as soon as the inflammatory process has extended to the peritoneal surface of the gall-bladder. The stomach, the duodenum or the colon may become attached to the gall-bladder either in acute or chronic cholecystitis. After stone formation has occurred, adhesions may develop at the site of an ulcerative process which permits the extrusion of a gall-stone or stones from the gall-bladder into the intestine. A number of cases of this kind have been observed in which the stone has, because of its size, caused intestinal obstruction. In the very acute gall-bladder infections with obstruction of the cystic duct, marked hydrops and phlegmon or gangrene of the gall-bladder, peritonitis frequently develops.

Severe inflammation of the peritoneum may result from imperfection of technic at operations upon the biliary passages; hence the greatest care must be observed for its protection during such manipulations. A number of observations have been made to demonstrate the fact that healthy bile is sterile and contains no bacteria. The truth of this proposition may be admitted, yet how can it be known in a given case whether the bile is healthy. Admitting that the bile in a traumatic rupture is healthy when the injury occurs it is true that it may not remain so, hence steps must be taken to close such a leak promptly.

The peritoneum, fortunately, is able to handle a considerable bacterial flora, and it has been shown that badly inflamed and thickened gall-bladders may be safely left when a drainage tube is inserted into the lumen to take away the infected bile. In cases of excision of the gall-bladder for necrosis or high-grade inflammations a drain carried to the stump of the gall-bladder or into the common duct takes care of the situation. Where for some reason a considerable portion of the gall-bladder remains following a partial excision, a drainage tube alongside usually suffices, because the peritoneum at once builds up a protective channel about such tubes and conducts the infectious material and bile to the surface. In case the external vent is not patent and tension occurs, a leak into the peritoneal sac may take place.

In chronic inflammation of the gall-bladder, when infection is absent, it is quite safe to excise the gall-bladder and leave the stump without drainage. It is also safe to suture the drainage tube into the gall-bladder by a double purse-string suture in cholecystostomy without attaching it to the abdominal wall. It appears that attaching the gall-bladder to the wall is a very safe measure, although some claim it leaves undesirable adhesions.

We are firmly impressed with the dangers resulting from infections of the bile passages in gall-stone disease and believe that life will be prolonged where drainage is employed. This, however, is a matter of personal opinion and men of wide experience believe the routine extirpation of the gall-bladder is desirable.

At operation for gall-stones or other affections of the gall-bladder, the greatest care for the protection of the peritoneal cavity should be exercised. The use of a suction pump to take up the fluid contents of the gall-bladder and prevent contamination of the cavity is of great value. The operative field should also be thoroughly protected by gauze pads. A most careful search must be made to see that all gall-stones are removed. This is done to prevent future symptoms and a secondary operation with its coincident dangers.

Operations upon the gall-bladder should not be deferred after the diagnosis of gall-stones is made. The longer such treatment is delayed the more numerous will be the adhesions and the more the function of the gall-bladder, the stomach, the duodenum and the pancreas will be crippled. There is always present the remote danger of development of carcinoma of this organ.

A very important portion of this study is to establish a diagnosis of cholecystitis and particularly the presence of gall-stones. Acute cholecystitis usually appears suddenly with perhaps a history of some previous dyspeptic symptoms. The attack may begin suddenly in the night with pain, nausea, and vomiting. The conjunctiva may be barely tinged with bilirubin, the urine may show a slight trace of bile. These symptoms are usually but not necessarily always present. Usually the temperature is elevated, sometimes as high as 103 degrees. Tenderness over the gall-bladder region will be well marked. Some abdominal rigidity will be observed.

In most cases, because of distention of the gall-bladder resulting from cystic duct obstruction, a palpable mass may be felt in the right hypochondrium. The bowels are constipated and the stools pasty and pale in color.

The attack may last for two or three days, clearing up as soon as the

swelling in the walls of the gall-bladder subsides sufficiently to provide drainage through the ducts. Soreness will be complained of for several days. During this attack gall-stones may not be present. In some instances when the drainage is free and the intestinal tract is kept open, there may be no recurrence. If the cholecystitis has followed an attack of typhoid fever with the *Bacillus typhosus* remaining in the gall-bladder as sometimes happens in the typhoid carriers, or if the causative bacterial flora of other types remains within the gall-bladder, a chronic type of cholecystitis may continue.

Naunyn has called attention to the manner in which concretions are formed in such cases. Exfoliation of the epithelium takes place and bile salts are deposited about the nucleus. Following this there is gradual accretion and an increase in the size of the stones. Solitary stone formation may take place, or the stones may be multiple. When more than one stone is present they show smooth facets upon their surfaces.

In cases where a solitary stone is present it may lodge in the cystic duct and cause hydrops of the gall-bladder. In certain cases it acts as a ball valve and causes intermittent hydrops with perhaps attacks of colic. Sometimes the colic is severe. The addition of an acute infection with swelling of the ducts and acute hydrops usually excites a sharp attack of acute inflammation upon a chronic one. This may result in gangrene of the gall-bladder, rupture from overdilatation or the development of a localized peritonitis about this region. Repeated attacks of gall-stone colic, continued soreness or a distended gall-bladder, particularly if attended with jaundice of varying degree, make the diagnosis and call for operative intervention.

Infections of the biliary passages, cholangitis, hepatitis, pancreatitis, give more severe symptoms and operative intervention may become more urgent.

The ability to demonstrate the presence of gall-stones depends upon the character of the stones and to a degree upon the skill of the roentgenologist. Pure cholesterin stones may cast no shadow. By present day improved technic a very large percentage of stones may be shown. Recently studies have been made with certain dyes to outline the gall-bladder and determine patency of its ducts. The clinical evidence of gall-stones should be followed in preference to a negative roentgenogram since stones may occasionally not be shown.

Considerable importance has been given to duodenal lavage in the diagnosis as well as in the treatment of gall-bladder infections. The method will prove of some value in each of these uses.

Certain it is, however, that gall-stone disease should not be allowed

to continue in the vain hope that by any therapeutic measure more than a temporary amelioration of the symptoms may be accomplished. That such symptoms may be improved by treatment and the gall-stones remain quiescent with slight discomfort must be admitted. It is not, however, for the best interests of the patient.

Obstruction of the common duct calls for prompt relief since the destructive action of the bile upon the renal, hepatic, and pancreatic function is well recognized.

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CHAPTER VI

SPECIAL FORMS—BLOOD STREAM INFECTIONS

PUERPERAL PERITONITIS

Puerperal infection was one of the great causes of mortality from childbirth up to the time of Semmelweis, of Vienna, some thirty years ago. Holmes first discussed its contagious nature in 1842.

Semmelweis came to the conclusion that puerperal peritonitis and the other forms of puerperal sepsis were due in the large proportion of cases to bacteria carried to the lying-in woman by the attendant. He attempted to prove that in the practice of certain obstetricians who were doing general practice along with this class of cases could be traced one infection after another.

The profession of Vienna was so profoundly shocked by his statements that he was completely ostracized as one who was detracting from their professional ability and honor. It was only after a number of years that his ideas became generally adopted and some time later a monument was erected in his honor.

Based upon his memorable work and that of his followers, the occurrence of puerperal fever and of puerperal peritonitis has become very much less frequent than formerly. A few cases do occur here and there in which the exciting bacterial cause is not borne by the obstetrician or the nurse, because of the previous presence in the genital tract or blood of the individual of pathogenic bacteria. The lay public has become so well educated as to the source of this form of infection and of its rare occurrence, that when it does happen the unfortunate obstetrician who has such a case receives almost universal yet usually unmerited censure.

Puerperal peritonitis arises following labor at term or an abortion before term because of the contamination of some part of the raw genital tract with pathogenic bacteria. These bacteria may reach the damaged tissue from the patient herself, or they may be carried to the genital tract by the attendant, either physician, midwife, or nurse.

The bacteria most often causative are the staphylococcus, the streptococcus, the pneumococcus, the gonococcus and the colon bacillus. Such bacteria, as has been repeatedly shown, are often present in the vaginal secretion of the apparently healthy woman. Usually these individuals

develop an immunity at least partial to the pathogenic properties of these organisms while there is no broken tissue surface on which they may grow. When, however, the placenta is separated at term or before, leaving a considerable raw surface, and when large lacerations of the cervix, the vaginal wall, or the perineum occur, there is offered a fertile field for the growth of the ever present organisms.

Then, too, from pyogenic foci in other parts of the body the blood stream may carry a colony of microorganisms which localize at the site of least resistance, the damaged genital tract, and cause the development of this form of peritonitis. That this blood stream infection occurs as a frequent determining cause I do not believe, although I am willing to admit the possibility of its occurrence. The autogenous inoculation locally occurs more often.

To the attendant, however, one must look for a considerable number of infections of the genital tract, particularly in those neighborhoods in which the doctor treats everything that befalls human flesh and brings the babies as well. It has been established that attendance upon a case of erysipelas, or of scarlet fever, or of measles and then visiting a woman in labor or going from an infected delivery to a clean one is the most fertile source of this affection.

Fortunately, it is becoming recognized at the present time that obstetrics is really a surgical proceeding, and the proper handling of such cases needs a complete surgical training in asepsis. The delivery of a woman of child is a mechanical process and needs a mechanical skill and training in surgical cleanliness to produce the best results.

The reason why infections were not more frequent in private homes in the past was due to the fact that in sparsely settled communities pathogenic bacteria have little to thrive upon and become attenuated, while the women become inured to the conditions and develop a high resistance.

In populous communities the bacterial flora is much more prolific and more virile, while the resistance of urban mothers to infection is lessened. As civilization advances so too does the necessity for precautions against infectious diseases.

The pathology of puerperal peritonitis is quite like that from the same type of organisms occurring in the nulliparous woman. The raw surfaces in the uterus and other portions of the genital canal make for a higher ratio of contaminations. The large venous sinuses in the uterus filled with new-formed clots make a splendid nidus for a bacterial colony. The same is true of contamination through the fallopian tubes and also through cervical, vaginal, or perineal tears. Pathogenic bacteria may enter the peritoneal structures through any of these paths. They may also

enter from a previously inflamed tube or ovary, the infection in which may date from the impregnation.

The changes taking place in these structures from the presence of bacteria are similar to inflammatory conditions elsewhere. Because of the overgrowth of the uterus and its vessels incident to pregnancy these inflammatory changes are very active and rapid in their development. Some of these patients die from toxemia before pus formation can take place in the peritoneal sac. In other cases the inflammatory process begins in the lymph spaces of the pelvis and in the broad ligament and spreads from that point into the peritoneal cavity.

The onset of acute peritonitis of this type is quite sudden. The second or more often the third day after the delivery the patient is seized with a distinct rigor or has chilly sensations. The temperature rises rapidly to 103 or 104 degrees. There may be a cessation of the lochial discharge, or the discharge may be scanty. In some instances at this time there may be considerable odor to the discharge. This is usually attributed to contamination with putrefactive bacteria (sapremia). At this particular time it is difficult and at times impossible to say whether the symptoms are due to the presence of putrefactive bacteria alone and the rise in temperature is due to the absorption into the blood stream of the toxins formed in their growth, or whether the infection is of a more serious type. The first type may clear up promptly or in the presence of a mixed infection with pathogenic microorganisms a most serious form of sepsis may arise.

The bacterial colonies may enter the blood stream through the large venous sinuses of the uterus, either as individual organisms or in some instances as clumps of bacteria or as clots filled with bacteria. In the first instance the symptoms usually described as septicemia result; in the second those of pyemia present. These symptoms confuse the picture of the three forms of sepsis, and when present as a complication of either type adds very greatly to the gravity of the case.

The peritoneal symptoms occur as a rule with pelvic pain, soreness, tenderness, abdominal distention, and vomiting. The last mentioned symptom does not appear as early as it does in some of the other types of peritonitis. Usually it marks the onset of the spreading form of the affection and is accompanied in many instances by an adynamic ileus. The "still" belly is quite characteristic. The pain and tenderness are pelvic at first, later may be elicited in the abdomen. This fact is important in the diagnosis. Boggy masses will soon be felt above the pelvic brim on palpation. The uterus becomes fixed. A boggy mass presents in the culdesac and in each broad ligament. These organs are very tender. The

uterus is patent, the os soft, discharging some thin serosanguineous pus or perhaps there is no discharge at all. There may be a markedly foul odor to the discharge or no odor at all. In the most grave types odor is absent. When blood clots or secundines are retained the odor may be putrid.

The symptoms just described may precede the symptoms of peritonitis by some hours. The diagnosis of puerperal contamination of some type is made immediately upon onset of the chill or chilly sensations and a change in the character of the discharge. It must not be forgotten that at about the third day an elevation of temperature occurs at the onset of lactation. This subsides promptly and is not accompanied by the pelvic symptoms described above.

To distinguish the character of contamination a culture taken from the uterus is useful. In well-regulated hospitals it may be well to take a culture from the vagina before and again at the completion of labor. In the cases showing serious symptoms one or more blood cultures should be taken immediately after the onset of suspicious symptoms. This is important to determine whether the blood stream is infected or whether the symptoms are due entirely to a local lesion or to a spreading peritoneal lesion which may later result in a blood stream infection.

The diagnostic evidences of puerperal pelvic peritonitis consist in pelvic pain, tenderness, backache, sensation of weight and heaviness, purulent vaginal discharge with or without odor, elevation of temperature and some depression. The mentality is clear as a rule.

The onset of the diffuse type of general peritonitis follows the above picture with the addition of vomiting, abdominal distention, general tenderness and rigidity, absence of signs of peristalsis, marked prostration, perhaps cold clammy sweating, and an anxious countenance.

It is not always possible to delimit puerperal peritonitis from puerperal sepsis with blood stream infection. In some cases of peritonitis the blood stream infection is an important part of the picture.

The extreme gravity of postpuerperal infective lesions makes the proper care of the parturient woman one of great responsibility. Many such cases come to labor without making proper provision for competent aid at the delivery, depending upon the hurried summons to the physician or the midwife.

No prenatal observation, no adequate aseptic precautions can be obtained. The surroundings are often bad under these circumstances.

Much criticism would be heaped upon a physician who would refuse to respond to a call for aid by a woman in travail. Yet it is unfair to ask a physician to assume the risk of professional ruin when he has no control over the circumstances under which he works. It is not within

the scope of this monograph to deal with these problems or with puerperal fever except as incidental to the subject of peritonitis occurring at this time.

We recognize the possibility of a pelvic infection occurring at the time of impregnation, leaving an inflamed tube or ovary. This may rupture during labor or be exaggerated thereby and produce an infection of the peritoneum. Such an infection cannot entirely be provided against except by a thorough pelvic examination before labor begins.

It is well known, however, that with the exception of the above state of affairs and that of a latent blood stream infection all other contamination of the genital tract may be avoided. This is true also of the bacterial flora present in the genital tract itself.

In order to accomplish such a happy result in a given pregnancy, the surroundings must be ideal. Because of this fact the parturient woman should when possible be delivered in a well equipped lying-in hospital. By this I do not mean a moderately clean boarding home, but a meticulously clean up-to-date maternity hospital, or department of a hospital, where every possible precaution may be taken.

For those women who are unable to provide these facilities the public hospitals should make the provision. These women should be under supervision long enough for a complete study of the physical condition. Their tonsils and teeth should be examined for focal infection, their renal function studied and the nitrogen content of the blood determined. Above all in importance the bacterial flora present in the vagina should be determined and if possible attenuated. The patient should be kept from all chance of infection from eruptive diseases. Under this régime puerperal peritonitis and sepsis cannot occur when the technic of delivery is carried out with absolute surgical thoroughness. The only chance for infection under these conditions lies within the pelvis of the patient herself. The records will show those microbes present in the outlet and provision may be made to care for them. The obstetrician can come to such a case with the absolute assurance that conditions are ideal for the prevention of infection and it is his duty to take his team to task for any break in technic, for upon him rests the entire responsibility.

Practically, then, puerperal infections should be classed as preventable diseases. Unfortunately, in civil practice such ideal conditions do not obtain and the efforts of the attendant must be directed toward obtaining the proper control of the patient and her surroundings during the latter months of gestation. In a very clean private home with funds to provide proper equipment and aid, a safe delivery may be accomplished. In the average home, however, the risks are great. Fortunately every woman

exposed to pathogenic bacteria does not have puerperal peritonitis. With the risk of being considered pedantic and tiresome, I will again insist that a thorough training in surgical asepsis is essential to the preparation for obstetrics. The absolute perfection of technic practically excludes the chance of contamination. Rubber gloves are essential and attention to every detail imperative. For those cases which do not have such ideal treatment, there must be provided some plan by which the peritonitis so very deadly in its results may be mitigated.

The best methods of dealing with this affection have perhaps not been fully settled, but whenever the lying-in woman becomes ill suspicion must rest upon infection until the actual condition is cleared up.

Placental fragments or blood clots should not be left in utero; but when they are present and cause symptoms what is to be done? Under the same exacting precautions the uterus must be freed of its contents. This must be accomplished with the greatest gentleness since the use of sharp instruments opening up closed venous channels may permit the passage of an overwhelming dose of bacteria into the blood stream. Personally the sharp curet, or even the blunt type, does not appeal to me for this purpose. The placental forceps can obtain the same results with less damage. A well dilated cervix permitting the gloved finger to enter the body will enable one to remove the larger fragments. Strips of gauze gently passed into the uterus will bring out the detritus.

Occasionally we have found benefit from a constant irrigation in saphrophytic types of contamination, but for the most part this has been abandoned for some years for rather cogent reasons. The conclusion has been reached that in all other contaminations and particularly in puerperal peritonitis no fluids save perhaps iodine solution should go into the uterus in these cases.

Deaver is strong in his statement that whenever a curettage is done the abdomen should be opened. In cases of puerperal peritonitis where the blood stream infection is not present it often becomes important to determine whether the expectant plan is to be followed or whether the radical plan of surgical intervention.

Undoubtedly there are some cases where the patient is so seriously stricken that any surgical intervention will clearly prove fatal. These cases should be treated expectantly even to the point of not opening the uterus or cleaning it out. By the use of the methods described under general peritonitis such cases may occasionally be brought to the operable stage or more rarely through the illness safely without intervention. Other cases show such marked local symptoms, with pointing of a suppurative process in the culdesac, whose condition precludes

an abdominal section. Yet they may be much benefited by a vaginal section. Still others, seen early, yet with a high-grade infection and considerable bodily resistance, may be carried safely through an abdominal section. When the indications are clear such treatment may be employed. The radical removal of the inflamed adnexa and even of the uterus itself to prevent a blood stream infection is sound surgery and will often bring these patients through safely and with the least suffering and the minimum loss of time. Cases which do not come under this class must be treated expectantly and in many cases come to recovery after many weeks, often remaining more or less incapacitated thereafter.

GONOCOCCIC PERITONITIS

Infection of the peritoneum with the gonococcus of Neisser is by no means a rare condition. It presents two varieties, acute and chronic.

In the first group of cases the bacterial invasion takes place suddenly, passing through a patent fallopian tube which has not yet been sealed by the swelling incident to the infection. A violent active gonorrhea is always present, the vulva being congested, covered with foul discharge, the vagina highly inflamed, and the uterine cervix soft, a thick mucopus exuding from the os. The vulvovaginal glands may be swollen, discharging pure pus. Instrumentation in such a case or even the application of iodine or other medicinal agent to the cervix may excite the spread of this disease to the peritoneum. Unfortunately this loathsome disease is often communicated to lovely, clean young women in the nuptial couch. Proper education in sex sanitation and a closer restriction upon marriage will markedly lessen this form of disease.

Promptly upon the entrance of a virulent strain of the neisserian organism into the peritoneal cavity a marked local reaction occurs. The tubes are engorged and bright red. Some lymph may be found on the surface and at the fimbriæ. The adjacent peritoneum becomes glazed and dry at first, but is soon covered with a thin layer of lymph. The adjacent ovary is similarly affected. In a short time the peritoneum is bathed in a thin serosanguinolent pus, which carries the infection rapidly through the general peritoneal cavity. A culture taken at this time will show a pure culture of the gonococcus. The absorption of the toxins from the abdomen is rapid, and they produce a marked systemic reaction, with evidence of involvement of one or more joints in some cases—first from the toxins and later from the bacteria. In the abdomen agglutination of the peritoneal folds occurs with pocketing of the fluid between coils of the intestine. Pus formation is not marked at

this stage, but later, probably from contamination with colon bacilli, there are many pus pockets formed.

These pockets may remain small and either empty into the intestine, coalesce into a large accumulation or be taken care of by the processes of absorption and repair. They may become quite chronic in the later stages, considerable time elapsing before the parts are restored to normal.

Many adhesions form during the later stages which usually disappear, but in some cases persist permanently.

Chronic Form.—The symptoms of the milder type are preceded by those of gonorrhea, usually, but not necessarily, of a less severe grade than the acute form. This local infection may persist for some weeks or months, when the patient comes down with pelvic pain, soreness in the lower abdomen, backache, constipation, and a low form of fever. The vaginal discharge may be temporarily lessened, but the leukorrhea soon reappears. Under rest in bed, under mild treatment, or sometimes no treatment, the patient may be on her feet again, but complains of weight in the pelvis, tenderness on exertion, and tires easily. Constipation is marked, and pain at stool may cause complaint. Contraction of the bladder in micturition may cause pain, and frequency may also be present. Exacerbations of the symptoms are prone to occur both at periods and from intercourse.

Sometimes an acute peritonitis is excited by leakage from the tube. Instrumentation is likely to excite such a condition. Examination will show very little reaction at the vulva, but a thick tenacious mucopus will be flowing from the uterine os, and in most cases considerable vaginal discharge is present. On bimanual examination the uterus will be found tender and firmly fixed. On each side and posteriorly a boggy, tender mass may be felt, which will be quite tender, both to the vaginal touch and by palpation of the abdomen. A considerable mass can in most instances be felt above the pubes. This mass may grow smaller as the acute process subsides, and after the peritonitis becomes quiescent a chronic form of salpingitis persists.

In some cases the onset is less severe, the pathologic process being essentially a local one. In this group the tube becomes infected, but the swelling of the mucosa and early closure of the fimbriæ prevent entirely or permit of only a minimum soiling of the peritoneum. The intensity of the process limits itself to the oviducts and the tissues immediately adjacent.

The walls of the tube become markedly thickened. Within the lumen pus accumulates, causing distention. A plastic local peritonitis limits the

process, while the omentum caps the inflamed portion and prevents the contamination of the general peritoneal cavity. The adjacent intestines also become adherent and aid in protection.

Infection of the ovary may occur with the formation of ovarian abscess.

Following an acute infection, or upon the milder form, there ensues a subacute inflammatory process with subsidence upon rest and recumbency, exaggerated by exertion, by exposure of the surface to chilling, and by the menstrual period. This condition may undergo repair to the point of fair comfort, but usually these women become chronic invalids. In some cases during the exacerbation, rupture of the tube takes place with the development of an acute peritonitis, which, however, is infrequently as severe as the acute form mentioned above.

A large number of single births are due to this type of infection. Tubes which have become occluded as the result of this gonococcic infection rarely become patent subsequently.

Occasionally pregnancy occurs following double salpingitis of this type, showing that though infrequent complete repair is possible.

Symptoms.—*Acute Form.*—This affection is usually preceded for one week or more by a high grade vaginitis with copious vaginal discharge, swelling about the vulva, and excoriations of the skin due to the irritating nature of the discharge. There will also be complaint of frequent micturition with dysuria. These symptoms are accompanied by sense of burning and heaviness in the pelvis. When invasion of the peritoneum takes place the patient suffers from sharp pain in the pelvis and abdomen, accompanied by a chill and a sharp rise of temperature, often as much as 103° F.

The abdomen becomes very tender, accompanied by distention and rigidity. Vomiting is a prominent symptom, and constipation is usual. In a short time pain may be noted in one or more of the joints, and may be distressing at times. The symptoms are quite similar to other forms of acute peritonitis, differing only in the mode of entrance of the invading organism and the location of its entrance.

The microscopic examination of the vaginal discharge will confirm the gonococcic nature of the affection.

Diagnosis.—The diagnosis of this form of peritonitis can be made by the history of an active gonorrhea with sudden intra-abdominal pain, high fever and chill in the acute form, with tenderness on pelvic examination, and a positive smear of Gram-negative diplococci within the cells of the vaginal secretion.

The chronic or localized form is determined by the history, by

the vaginal discharge, by the pelvic and abdominal pain, by the fixation of the uterus and adnexa, by the boggy masses behind and on either side of the uterus, and by the fever.

Prognosis.—The prognosis of gonococcic peritonitis is always serious, but not so grave as some of the other forms in so far as an immediate mortality is concerned. The more acute highly toxic form is the most grave, and in some individuals with diminished resistance, or in cases following labor or abortion, or where a grave intercurrent trouble, as a renal, pulmonary, or cardiac lesion, or an acute pneumonia or pleurisy complicates the affection, the prognosis becomes exceedingly grave.

The milder types of the acute form and the chronic types of the disease are less grave, showing a lower mortality rate from operative than from nonoperative treatment, the latter being higher.

There is, however, a serious danger in these cases, since the unoperated cases almost always remain invalids, suffering from backache, pelvic pain, leukorrhea, constipation, and in many cases recurrent attacks, abdominal pain, and occasionally intestinal obstruction. Careful surgical management will in most cases restore these patients to comparative health.

The nervous phenomena incident to the complete removal of the adnexa are well known, and these patients require careful supervision to keep them in a fair state of mental equilibrium.

Treatment.—This will largely depend on the form. The usual treatment for the acute form will consist of complete rest in bed in the semi-sitting or Fowler position, with the local application of ice to the abdomen and the use of normal saline solution with one or two drams of bicarbonate of sodium given by proctoclysis with the Murphy drip method. In some cases the patient complains of the cold, and the use of the hot water bag may be substituted for ice. Pain should be relieved by opiates. Nothing should be administered by the mouth until nausea subsides. After such time water may be taken and liquid food when it causes no distress. For the severe nausea and vomiting sometimes seen, nothing equals gastric lavage. Purgatives should not be given until the acuteness of the process has subsided. The lower bowel may be emptied by enemata. When the temperature subsides and the local reaction becomes quiescent, if the patient's condition justifies it, the pathology should be cleaned up by abdominal section. Often the patient's resistance will be improved by some weeks' delay during which the process becomes localized and the pus accumulations in the pelvis lose their virulence.

Some surgeons are much more radical in their treatment of these cases, and advocate immediate operation in acute gonococcic peritonitis, claiming an equally low mortality, with a marked economic saving by this method. It appears to the writer that this is a point to be determined largely by the individual, based upon his own results. There is probably a small increase in the primary mortality in the cases treated by early operation, which may be overbalanced by the later complications and the morbidity incident to expectant methods.

For the localized or chronic form, similar methods are in vogue, but this class of cases will be more safely carried through the active process than the acute type. There are certain cases which have improved, but in which sharp symptoms present, indicating a leaking tube which should be handled by immediate operation. In these patients the resistance has been built up, and by prompt removal of the inflamed adnexa, a spreading peritonitis is prevented and prompt recovery assured.

The operation usually employed in acute gonococcic infections consists of a median section about 4 inches (9 cm.) in length after proper skin preparation, under general anesthesia. The operative field should be protected from the fluids escaping from the peritoneal cavity, by draping the raw wound surfaces with several thicknesses of gauze or with towels. In addition an automatic pump takes up the escaping fluid promptly without contamination of the walls or loss of time. While it has been clearly demonstrated that it is impossible to cleanse the peritoneal cavity of the infectious agent in any of the peritonitides, yet it is undoubtedly a fact that in a case of this type where a free culture of the causative organism can be obtained, it is quite feasible to remove by suction the overload. Under these conditions the peritoneum will in all probability be able through its own protective forces to take care of the remaining infection, aided by such minor assistance as is provided by proctoclysis, by gastric lavage, nourishment at the proper time and the customary methods generally employed. Usually the removal of both the infected tubes and ovaries will give the best results, doing away with the necessity for subsequent operation for the relief of pyosalpinx and ovarian abscess. In some instances, when the condition of the tubes and ovaries seems to justify an effort to preserve their function, it may be justifiable to leave them intact, or to leave one intact when possible.

Some have recently advised splitting the tube longitudinally to avoid the constrictions which form subsequently and prevent conception. The efficacy of this procedure is not yet established. When the ovary is not damaged or there is not much evidence of disease in the early acute

cases, it should be preserved. In the chronic cases the preservation of any healthy portions of this organ is desired. It is not considered justifiable surgery to perform an incomplete operation and leave markedly suspicious or badly infected tissue with false hope that the patient will recover. Such cases remain more or less permanent invalids, and the surgeon must carefully weigh the advantages of each plan before proceeding to the completion of the operation.

Under the subject of drainage will be discussed more fully the indications and contraindications for its employment. In this type of infection, the majority of patients will do well without drainage. In the high grade acute infections of this type, a few cases will do better if drained. If the adnexa are left in situ in such cases the coffer-dam or a large soft rubber-gauze cigarette drain may be employed to assist in removing the possibility of tubal infection. The usual advice is to do without it if you can.

In the chronic localized lesions which have existed for some weeks, the virulence having subsided, drainage usually is not needed. Very ragged tissues remaining with considerable damage to the endothelial structure may call for a drain.

PNEUMOCOCCIC PERITONITIS

This type of acute peritonitis is thought to be the result of blood stream infection. There is a possibility of invasion from the pleural cavity, but this seems remote when one considers the direction of the lymph channels and the distribution of the lymph vessels.

It is established, however, that retroperitoneal inflammations may contaminate the pleura or the lungs. Abscesses forming in the abdomen have been known to open through a bronchus. Therefore, it is within the range of possibility that infections in the mediastinum or lungs might be conveyed to the peritoneum.

The peritoneum may be the only serous membrane affected. All of the serous membranes may be simultaneously affected, however.

Pneumococcic peritonitis occurs more often in children than in adults, and in female than in male children. It is quite likely from this observation that the infection may enter through the genital tract and the fallopian tubes.

There may be a recognizable focus in the lungs or the middle ear, from which the infection is derived. The peritonitis may arise prior to the above lesions. The tonsil may be the source of infection.

Predisposing or Contributing Causes.—Among these factors

may be mentioned season of the year, since during the winter months all pulmonary and allied affections are more frequent.

A more certain contributing cause is the congregating of many persons together in crowded, ill ventilated rooms because of the cold weather. This leads to prolific bacterial growth and a marked increase in their virulence.

All forms of debilitating illnesses tend to favor the development of this affection. It is associated most frequently with the respiratory diseases as bronchitis, pneumonia, pleurisy, and empyema. Middle ear disease, meningitis, endocarditis are present in a considerable proportion. They are usually present in blood stream infection. Inflammation of certain glandular structures also results from the blood contamination. Parotitis, thyroiditis, orchitis, osteomyelitis, one or more may be present.

The causative factor can be established by careful bacterial examination. Cultures should be taken from the peritoneal sac and from the blood also in all cases where the latter is likely to be invaded by the organism. Upon the laboratory findings to a considerable degree will depend the treatment. The type of organism present has a bearing, particularly upon the employment of serum in the treatment.

This affection is not of very frequent occurrence if the small number of cases recorded in the literature is taken as a criterion, but it probably occurs more frequently than the reported cases seem to indicate. It is quite likely that a large number of cases are overlooked as far as the causative agent is concerned, due to the failure of many surgeons to take bacterial cultures of all intraperitoneal fluids. This does not seem improbable since it is only within recent years that properly equipped laboratories have been added to the facilities of many hospitals.

It seems evident that the study of this form of peritoneal infection has received more attention from the French than it has from American or English writers, since there is a great preponderance of French contributors upon the subject.

It has been suggested that climatic and racial conditions and mode of life may be such that this form of infection perhaps occurs more often in France than in America. However, no evidence has been submitted in support of this suggestion.

The number of recorded cases is not sufficient to formulate any fixed and definite statements in regard to the affection. According to Hertzler 160 cases have been recorded in the literature upon this subject.⁵ He says that many of these cases have not been positively established as cases of pneumococcic peritonitis. He properly holds that

merely finding a diplococcus in such an inflammation scarcely rises to the dignity of a scientific proof of fact.

He mentions 17 cases reported in America as authentic, 2 by Flexner,⁴ 6 by Pearce,¹⁰ 1 by Stone,¹¹ 3 by Woolsey,¹² and 5 by Mathews.⁷

The difficulty in positively identifying the causative organism is because of the difficulty in obtaining its growth in culture media. The bare fact that a pneumococcus is present in the peritoneal fluid in pure culture is strong presumptive but not positive evidence of its causative relation to the disease. There is this fact in favor of its being the actual cause when the culture is pure, namely: the pneumococcus is usually overgrown by most other pathogenic microorganisms. This statement was abundantly proved by the work in the study of empyema in the army hospitals during the World War. If any other colonies had been present at the inception of the disease they would almost certainly have remained or probably would have overgrown the pneumococcus.

Reasoning from the above premise one must accept as a causative bacterial agent pure strains of pneumococcus when no other organisms are found in acute peritonitis.

It does not appear to be necessary that a patient should develop pneumonia in order to have this type of peritonitis. In fact, it does not appear that the relationship is very close since in the literature only about one per cent of cases of pneumonia shows a complication of peritonitis. On the other hand it is quite likely that almost as large a number of peritonitis cases will show at least one per cent of cases of pneumonia as a complication. That these pneumonias complicating peritonitis are due to pure cultures of pneumococci does not seem probable since many of them are due to other organisms. A considerable number are caused by aspiration of secretion from the throat. Some are the result of etherization.

The cases coming particularly under the head of pneumococcus peritonitis are in many instances due to a direct contamination of the peritoneum from pneumococci which have entered through the intestinal or vaginal tracts.

Another group finds the organism entering through the throat, tonsil, or middle ear. Again, there is a possibility of the invasion of the lungs through the air passages and transportation thence to the peritoneum. This transportation may occur, according to some, through the lymph channels and the mediastinal glands. The transportation from the lungs to the peritoneum may occur through a blood stream infection. In fact Michaut,⁸ Desquin,³ von Brunn,¹ and others claim that all cases of pneumococcic peritonitis are of this type and the peri-

tonitis is only a part of the septicemic process. While it must be admitted that this method of invasion does occur it does not necessarily follow that all cases so arise.

I have personally observed the transmission of pneumonia from one patient to another by contamination of the food upon the table carried there by flies. This observation indicates the entrance of these organisms into the patient's system by the intestinal route, also the transportation therefrom to the lung. This evidences the fact that the germs may enter the intestine and presupposes the possibility of contamination of the peritoneum. It also indicates the certainty of blood stream infection from infection through the intestine.

Jensen ⁶ attempted to establish a hematogenous pneumococcic peritonitis by way of the intestinal tract. He fed young rabbits, without previous intestinal lesions, virulent bouillon cultures of streptococci and secured a peritonitis, and Griefswald got the same result with pneumococci grown on milk.

"The source of the infection from the mouth via the intestinal tract is easily hypothecated, but satisfactory proof of its occurrence has not been provided." ⁵

It appears that no matter what the point of invasion there is always possible a blood stream infection. Proof of this is demonstrable by cultures from the blood and a little professional attention to this point should soon clarify the situation.

Hertzler ⁵ makes the statement that as yet no case has been proved to be of hematogenous origin, and quotes Zesas as stating that no case of peritoneal infection has ever been produced experimentally by injecting organisms into the blood stream.

Some of the grave cases of peritonitis, pleurisy, meningitis, and pneumonia combined are due to a mixed blood stream infection.

The contention of some that the greater frequency of its occurrence in females, particularly young girls, indicates the contamination through the fallopian tube has not been fully accepted, but a number of cases have been recorded in which pneumococcic salpingitis was observed.

Pearce ¹⁰ reports rupture of such a tube and the development of peritonitis therefrom. Pearce also reports two cases in which the pneumococcus could be demonstrated in the endometrium.

Patellani Rosa ⁹ reports 13 cases of pneumococcic salpingitis in 945 cases of tubal disease.

Peritonitis has been caused by pneumococcus infection of the gall-bladder and of the urinary bladder as well. The infection atrium was not determined.

The question has not been determined whether the intestine permits the passage of the pneumococcus more readily than other forms of bacteria or not. Nevertheless, it appears that the pneumococcus does pass. Many reports give evidence of its causative action by invasion through the appendix. It also seems evident that the pneumococcus must have some selective affinity for the serous membranes as well as the pulmonary structures.

Pathology.—This type of organism is perhaps a slower pus producer than some other bacteria and there is excited by its presence within the peritoneum an abundant fibrous exudate. Because of this fact there is more or less tendency to sacculation and many localized abscesses may form.

Because of these facts this form of peritonitis runs a mild course. The exudate is usually abundant with a considerable amount of fibrin production. From this action many firm fibrous adhesions are formed. The disease is often localized and sometimes the rapid increase in amount of the exudate pushes unaffected areas to one side. An abscess may be entirely surrounded by adherent intestines. The agglutination is so marked that the entire mass of intestines and abscess may come out together. These abscesses show thick, creamy pus from which the causative organism may be removed. Some, however, may be sterile.

These abscesses tend to center near the umbilicus and occasionally protrude or even rupture at this point. It would seem likely, if the invasion occurs through the fallopian tubes, that the abscess would be located in that neighborhood. The fact that few of the abscesses locate near the diaphragm seems to indicate that infection from the pleura and mediastinum is not very frequent.

Certain other lesions are present frequently in this type of infection. The most notable of these are middle ear inflammations, pleural and pulmonary inflammatory processes. These may either precede or follow the peritoneal affection.

Abscesses in the liver may be looked upon as complications. Meningitis may be an integral part of a general bacterial infection or a complication of the peritonitis.

Some of these cases in which the bacterial poison is very intense begin as general peritonitis, while others are more or less localized from the onset.

The severer forms show a marked toxemia and blood cultures may give positive results. In the more localized types this finding will probably not be present.

The symptoms of the acute generalized form closely resemble acute

perforative peritonitis. There will be observed pain, vomiting, nausea, a rigid abdomen which in the early course of the disease will be contracted or scaphoid in appearance.

The impression seems to be general that these cases are ordinarily mild, but when we consider that in a number of them the pleura and the meninges as well as the peritoneum are involved, we cannot encourage this belief. Some of these cases are extremely severe and the prostration is so marked that the outcome is grave. Blood stream infection with high grade pleurisy, particularly if pneumonia be present, usually terminates fatally. The milder forms where the process remains localized show the usual symptoms of this type of peritonitis from other causes.

Pain, vomiting, localized tenderness and rigidity, malaise, elevation of the temperature, abdominal distention somewhat localized with a palpable mass, differentiate this form of localized peritonitis.

Pain is usually intense at first and is generalized. Later it becomes less severe and is localized. Vomiting is perhaps more constant than in some of the other forms of peritonitis. It presents early and continues for some days. One of the most characteristic symptoms is diarrhea. The temperature at the onset may be high, similar to that in croupous pneumonia. It may have a sudden drop, also similar to that occurring in pneumonia. An excessive toxemia or blood stream infection may produce a decided fall in temperature. The pulse does not accurately follow the temperature, but it does the prostration.

In young children the onset may occur with a convulsion, while a chill may usher in the symptoms in an older person. Headache is of frequent occurrence, and may be so severe as to make the suspicion of meningitis strong. Meningitis may in fact be present. Herpes is frequently present in these cases; an important observation, since it is infrequent in other forms of peritonitis.

Some of these patients, presenting pain, tenderness, and rigidity, clearly indicative of peritonitis, do not appear as ill as these symptoms from other forms of peritonitis would make them.

According to Comby and Grancher the temperature may end by crisis. Usually there is a gradual fall as the symptoms improve. These cases may be less stormy in their symptoms than the other types of peritonitis, but they are more likely to persist and they progress more slowly to a final recovery.

Rupture of the abscesses which may form in this affection occasionally occurs either at the navel, in Scarpa's triangle, rarely into the intestine, which seems strange in view of its close proximity.

When the abscess persists the patient becomes emaciated, the temperature ranges high and is intermittent with sweats and may be mistaken for tuberculosis. The diffuse type is seen in adults; the localized in children.

Diagnosis.—The diagnosis of this affection is based upon the sudden onset, pain, nausea, vomiting, localized distention, tenderness, elevated temperature, and diarrhea. There may such marked headache as to cause suspicion of typhoid fever. The absence of the spots of Louis or the mental hebetude and a negative agglutination test for typhoid exclude the latter.

There is a very close resemblance between the acute form and appendicitis. In fact the infection may appear in this organ. When it develops elsewhere the local symptoms will distinguish it. Gonococcic peritonitis may be differentiated from the pneumococcic form by the different staining qualities of the organism, and by the severe preceding local infection with its irritating discharge. Tuberculous peritonitis in the large majority of cases runs a much slower course.

There is a large serous accumulation in a large percentage of cases.

Treatment.—By proper hygienic measures, ventilation of schools, churches, moving picture houses, theaters, and homes, much may be done to prevent this as well as the respiratory and eruptive diseases.

At the onset of the disease the patient should be put to bed and examined very carefully. It may be possible that other lesions may obscure the peritoneal symptoms. When the latter are prominent and a diagnosis of peritonitis is made, operation will probably be performed at once. The type of bacterial flora may not be recognized prior to the section. If the primary site of infection is in the peritoneum, this mistake will not be harmful but beneficial. The perforative leak will be found perhaps at the appendix, gall-bladder or tubes and the further entrance of bacteria checked.

It seems likely from our present knowledge that when the blood stream is known to be infected, as in the presence of endocarditis, it will be safer to defer abdominal section. If the patient has a pneumonia and this is typed, the early use of pneumococcic serum for this particular type of organism is indicated. The results are striking in some groups, entirely negative in others.

The more recent work of Young and others in the use of intravenous injections of two per cent solutions of mercurochrome, and the use of gentian violet for blood stream infection opens up a wide field for the-

rapeutic investigation. The intravenous instillation of hexymethylamin may also be worthy of a trial.

Prior to operation, the local measures generally employed in peritonitis are useful. For nausea and vomiting gastric lavage should be used. For support, rectal instillation of normal saline solutions containing glucose, sodium bicarbonate and adrenalin solution (1:1000) are beneficial. Ice applied to the abdomen will relieve pain. It may also be useful over the heart in blood stream infections. Morphin may be employed with benefit to relieve pain and produce sleep.

With subsidence of the acuteness of the process, careful feeding and tonic treatment will aid in carrying the patient through. Careful consideration should be given to the determination of the best time for intervention. The proper procedure depends upon the findings at operation. Some small circumscribed abscesses which can be delivered in toto through the wound may be excised en bloc. This may entail the excision of an intestinal loop, but the convalescence will be less severe and recovery more rapid by this method. The serious nature of resection should be carefully weighed before this step is undertaken. In other cases the emptying of the abscess cavity with separation of all adhesions will bring about prompt repair in most cases. When many ragged surfaces remain uncovered a drain will be necessary. Raw surfaces should be covered carefully, where possible, to prevent the formation of adhesions.

TUBERCULOUS PERITONITIS

Tuberculous peritonitis is the result of infection of the peritoneum with *Bacillus tuberculosis*. This infection may enter through the fallopian tube in the female (Fig. 36), through the intestinal wall or may be carried to the peritoneum either in the blood or lymph stream and there deposited just as it is in the bones or joints. The author has seen acute tuberculous peritonitis occur from rupture of a caseous lymph-node into the peritoneal cavity. It may occur as one of the lesions of general tuberculosis, for instance in an acute miliary tuberculosis or as a secondary deposit in the pulmonary, renal, or intestinal form of the affection, or from extension to this structure of an extraperitoneal lesion. While there are cases in which no other demonstrable lesion can be found it seems probable that outside of the local involvement from the intestine or adnexa there is always present at some point either an active or a healed tuberculous lesion prior to the development of the peritonitis. This local lesion often heals leaving to all intents and purposes the peritoneum as the sole structure actively involved in the process.

The peritoneum seems to be peculiarly susceptible to attack from the tubercle bacillus, and shows much less resistance to its progress than it does to pus producing organisms. The latter have been injected in pure culture into the peritoneal sac experimentally without exciting more than a local reaction, while the implantation of the minutest fragment of tuberculous tissue in susceptible animals almost universally results in localized and general tuberculosis. Upon this fact is based one of diagnostic tests of renal tuberculosis. Urine from a patient suspected of having a tuberculous lesion injected into the peritoneum of a healthy



FIG. 36.—ILLUSTRATING TUBERCULOUS SALPINGITIS.
Note the numerous small tubercles. (Author's case.)

guinea pig is promptly followed by a local and general tuberculosis if the bacillus is present, and is deemed conclusive evidence of renal tuberculosis.

After the peritoneal structure is invaded by the bacillus of Koch depending upon the dosage (number of bacteria), upon their activity, and upon the susceptibility of the patient to the particular type of infection, there occurs quite a variation in the pathological lesions resulting. Moreover, it seems quite likely that the human peritoneum, in not a few instances, is able to overcome a mild invasion with this type

of organism without the development of peritonitis. In yet other cases, after this form of peritonitis is well developed, the patient builds up an immunity which overcomes the infection and spontaneous recovery takes place. Again, in some rarer cases the presence of an acute suppurative infection may by the very activity of the inflammatory process excited, overcome the slower but more persistent tuberculosis, since it is a well known fact that the bacillus of tuberculosis does not thrive in the presence of an active pyogenic process. This is particularly true of the lymphatic structures. Per contra sometimes a mild pyogenic process may break down an already weakened resistance and permit a rapidly progressive development of tuberculosis.

Most bacteria invading a structure produce practically the same lesion in every instance. This is not the case with tuberculosis of the peritoneum, since the effects of its implantation are by no means uniform. Perhaps the most frequent lesion met is the multiple granular tuberculosis of the peritoneum. At the site of invasion one or more soft grayish masses (tubercles) form, and in a short time from the contact with the serous coat of the intestine as it moves about under respiratory excursions and from physiologic peristalsis, the entire surface of both visceral and parietal peritoneum becomes studded with the same type of soft polypoid masses, varying in size from a grain of rice to a small nut. These structures only infiltrate the gut wall for a short distance, and are not supplied with blood-vessels, hence there is always present a tendency to limitation of their size and extent. In cases where the walls of the gut become thickened so as to interfere with its mobility there is a tendency for these lesions to coalesce and result in adhesions between the intestinal coils with pocketing or sacculation of the serous fluid, and in this manner is developed the circumscribed or localized ascitic accumulations. By diminishing peristalsis and the formation of fibrous bands healing may take place even at this stage. In some cases this progresses until complete occlusion of the intestinal lumen results, which may be the determining factor in sending this patient to the surgeon. In those cases which progress to spontaneous recovery there is abundance of fibrous tissue formation. Much of this subsequently disappears as time goes on, and such an abdomen may show but little or no evidence either of the tubercles or the resulting fibrous bands when examined after repair is complete.

Charles N. Dowd reports a case which was under observation for an unusually long period and permitted a corresponding opportunity for studying the natural history of tuberculous peritonitis.

At the age of five, twenty-one years ago, she was admitted to St.



FIG. 37.—TUBERCULOUS PERITONITIS.

Showing typical tubercles and marked tuberculous thickening of the parietal peritoneum. Drawing made at autopsy of American soldier in France. (Case of Dr. John W. Moore.)

Mary's Hospital for children. Dr. Dowd operated for tuberculous peritonitis and found the peritoneum studded with myriads of tubercles and the omentum contracted into a thick mass. She had much ascites at this time.

First Operation.—The operation consisted of simple incision with exposure of the abdominal contents and the peritoneum to the trauma of the operation, with immediate closure. She was reasonably comfortable for some years. Eight years after her primary operation a sinus opened in the original abdominal incision, discharging for a week and closing spontaneously.

Between 1913 and 1916 she had considerable abdominal pain. In January, 1916, fifteen years after her primary operation, she was admitted to Roosevelt Hospital on account of attacks of vomiting and pain in the region of the appendix.

Second Operation.—The appendix was removed. It showed no gross lesion, but the walls were more thickened than normal, possibly two or three times the normal. This gave an opportunity of studying the condition of the peritoneum. There were no visible tubercles in the region where there had formerly been so many. There were a few adhesions about the uterine appendages. The vermiform appendix was covered by adhesions which attached it to the wall of the cecum. The caput coli was thickened so that it was not possible to bury the stump of the appendix in the ordinary way, but no active tubercles were present. The scar of the old incision was excised and the muscles there looked particularly strong and well nourished and the wound healed primarily. Examination of the microscopic section from the appendix and from the tissue of the abdominal scar showed no evidence of tubercle tissue.

She then continued reasonably well for about five years when she again came to the Roosevelt Hospital, September 30, 1921, on account of severe pain in the lower part of the abdomen. She had lost twenty pounds in the previous year.

Third Operation.—This operation, October 3, 1921, was for the relief of excessive paroxysms of pain in the lower part of the abdomen. At this operation the ovaries and tubes were found encased in inflammatory tissue and were removed excepting a small bit of the capsule of one ovary which looked normal and the base of one tube which seemed suitable for plastic repair. There was much inflammatory tissue about the uterine appendages and it was not possible to tell at that time whether the inflammation indicated fibrosed tuberculosis or some other type of inflammation. At this time further examination of the peritoneum was also possible. There was no evidence of tubercles on the

parietal or intestinal peritoneum or about the caput coli. Microscopical examination of the tissue removed showed bilateral tuberculous salpingitis. She is free from pain and feels well and has gained twenty-four pounds in the last year.

In certain other cases the protective function of the mesentery overcomes the beginning infection by encircling the local lesion, isolating it and walling it off from the general peritoneal cavity until the patient's tissue cells can overcome the disease. In yet other cases particularly those with a pulmonary lesion the patient's resistance is so much lowered that the lesions persist and increase until death ends the scene. If there is present an extensive tuberculosis of the intestinal wall, either primary or secondary to the peritoneal lesions, large masses of granulomatous tissue form, which rarely and in some young individuals may become quiescent or even undergo repair but in the larger number of instances progress steadily, either resulting in intestinal obstruction, local suppurative processes from bacterial invasion from the intestine, or in death from general tuberculosis or malnutrition.

The microscopic findings in tuberculous peritonitis vary with the type of the disease. In cases of general peritoneal involvement with multiple tubercles covering the serous surfaces and a quantity of free fluid in the abdomen, the picture is quite characteristic. Section through the intestine at the site of one of these lesions will show cellular infiltration into the serosa and the subserosa with thickening of both layers. The muscularis is not much involved. It only shows ulceration in those cases in which bimucous fistulas are formed. The tubercles themselves appear as collections of cells extending some distance above the surface of the peritoneum. These structures are nonvascular, contain large numbers of lymphoid cells, epithelioid cells and a few giant cells. The latter contain within their structure and lying about the periphery a number of tubercle bacilli. A very delicate reticulum can be demonstrated as supporting the cells. Owing to the avascular structure the center of such masses undergoes degeneration and caseation. Necrotic changes are seen in this location. About the base of the mass mitotic changes are observed in the connective tissue cells together with a number of lymphocytes. The fibroblasts are converted into fibrils and the connective tissue stroma is increased. New vascular structures are observed at this point. As the reparative process continues the new tissue contracts, the tuberculous structure softens and the necrotic material is absorbed.

In the plastic types of the disease the tubercle presents in large masses which may involve considerable areas of the peritoneum. On

section these masses show the picture of tubercles embedded in a large amount of new connective tissue. The cellular structure of the mass is similar to the one previously described. The mass itself is much thicker, more extensive, and contains but few bacilli. The large masses are mistaken occasionally for tumor formation. The omentum and the mesentery are alike involved. The retroperitoneal glands are greatly thickened and infiltrated with tubercles.

Forms.—There are two general types of tuberculous peritonitis, acute and chronic. The acute process occurs most often in younger individuals, as a result of a massive invasion of bacteria, as from degeneration and rupture of a caseous lymph node, from invasion through the fallopian tubes, or from rupture of a tube distended by the products of a tuberculous lesion, or from the rupture of a tuberculous appendicitis. This type of lesion results in a rapid involvement of the entire peritoneal structure with a marked general and local reaction. It may assume the type of general, circumscribed, or localized suppurative peritonitis. These lesions may result in rapid extension, especially when connected with advanced pulmonary lesions or in acute miliary tuberculosis, or the infection may subside into subacute or chronic peritoneal tuberculosis. The cases in which the abdominal fluid consists of a clear or very slightly flocculent serum will be most likely to become subacute or chronic. The presence of this serum tends to keep the coils of intestine apart, and, as a rule, intestinal obstruction does not develop in connection with these cases until the process begins to subside and is less frequently seen than in the fibrinous or serofibrinous type. This fluid does not show the causative organism on microscopic examination, and cultures taken from it remain sterile, probably because the bacterium is embedded in the granuloma at its attachment to the intestinal wall. While acting as a protection against intestinal adhesions this ascitic fluid seems to favor the progress of the infection, since its removal by abdominal section with exposure of the cavity to the air even for a short period seems to favor markedly the reparative process. I am firmly convinced that this is a clinical fact. Sir Spencer Wells accidentally discovered that this simple procedure was beneficial in cases of tuberculosis of the peritoneum with ascites.

Chronic tuberculous peritonitis differs from the acute form more in degree than in kind. The lesions are similar, though perhaps not so extensive, and persist through many months or even years. The lesions are fibrinous, fibrinoplastic or of a dry type in some cases. In others they are suppurative, and in yet others they appear as multiple fine granulomata or tubercles bathed in a large quantity of serous fluid.

Therefore, the following terms have been applied to tuberculous peritonitis:

1. Tuberculous ascites
2. Fibrinoplastic peritonitis
3. Adhesive peritonitis
4. Suppurative or caseous tuberculous peritonitis, or mixed infection.

The clinical history of these lesions usually persists through considerable periods of time. Recent investigations seem to show that persistence of local tuberculous lesions about the appendix or about the uterine appendages in the female tends to keep up the process. Undoubtedly reinfection from the intestine from pulmonary lesions also tends to cause the persistence of the lesions, for it seems established that cutting off the entrance of bacteria tends to limit the process, which, like all tuberculous processes, tends to recovery unless the patient's resistance is overloaded. Many cases are on record where the patient was progressively losing ground, and the removal of a local lesion, say an appendix or infected uterine appendage, has been followed by prompt and often by permanent cure.

The acute form may begin with sudden intra-abdominal pain which may be mistaken for an acute appendicitis or an acute salpingitis of infective type. This is particularly true of cases developing from rupture of a caseous lymph-node. In such cases the temperature will probably run a higher course than most cases of acute appendicitis. The abdomen will be tender, some rigidity will be noted, more often on both sides, and in some instances the pain will be greater upon the left side, particularly if the left ovarian tube is the site of the infection. There will usually be but little nausea or vomiting noted. Constipation may be a marked symptom. The blood picture will not show the marked leukocytosis noted in suppurative processes, and is a valuable diagnostic sign. In a short time the presence of ascitic fluid in the abdomen can be demonstrated, which will aid in differentiation. The typical afternoon rise of temperature and its persistence are somewhat characteristic of tuberculosis.

The chronic form of tuberculous peritonitis begins gradually. In the plastic type its development may be so insidious that the patient first takes notice of his trouble when intestinal obstruction presents. The hydropic or serous form begins very slowly. The patient, if a child, shows gradual enlargement of the abdomen, with a disinclination to play, perhaps with little or no fever, and his appetite may or may not be impaired. In a short time there will be loss of weight, pallor, and shortness of breath, due to the pressure of the accumulated fluid.

The abdomen is doughy to feel and protrudes markedly at the navel. Often at first examination early in its development this symptom is attributed to worms.

In the adult the ascitic form usually runs a chronic course, developing slowly with anemia, some loss of flesh, enlargement of the abdomen and a slight or no afternoon rise of temperature. There may be little



FIG. 38.—TUBERCULOUS ASCITES.

apparent change in the general health, but close questioning will reveal the fact that the patient tires on slight exertion and is, in fact, below par. A careful physical examination will elicit the presence of free fluid in the abdomen. This is evidenced by flatness in the loins upon recumbency, the dulness changing with the position of the patient. In the erect or sitting posture the fluid accumulates in the lower part of the abdomen, while the crowding of the intestines upward gives a tympanitic percussion note to this region.

Localized or encysted ascitic accumulations do not give these changes upon percussion, and there is found an area of dullness surrounded by tympany. Also an irregular mass may be palpated.

Both of these findings simulate closely the physical signs of a proliferating ovarian cystoma. Careful examination of the uterus and adnexa should enable one to arrive at a correct diagnosis. Pain is not an important factor in these cases, neither is tenderness at all constant. Both these symptoms are moderate in degree when present.

The presence of free fluid in the abdomen should arouse a suspicion of tuberculosis when the usual causes of ascites, cirrhosis of the liver, valvular disease of the heart, or an intra-abdominal malignant growth can be excluded. In ascites due to renal disease the accompanying dropsy of the limbs, anasarca, with the urinary findings will give positive diagnostic signs (Fig. 38).

Diagnosis.—The diagnosis of tuberculous ascites is made by the presence of an increasing ascites, in which the fluid changes its site with the position of the patient, by the slight loss of flesh, by the loss of strength, by the slight evening rise of temperature, the moderate severity of symptoms, a positive tuberculin reaction, and by the exclusion of the following conditions which might be mistaken for it, *viz.*:

- Cirrhosis of the liver
- Carcinoma of the liver
- Cyst of the pancreas
- Splenomegaly
- Banti's disease
- Ovarian cyst
- Distention of the gall-bladder
- Pyosalpinx
- Hydrosalpinx
- Pregnancy
- Ectopic gestation

The plastic type of peritoneal tuberculosis is not accompanied by effusion into the peritoneal cavity. The bacillus in this type excites an active reaction in the tissue cells, and the inflammatory product consists of marked cell proliferation with formation of gelatinous fibrin, which agglutinates the adjacent serous surfaces, fixing the intestines in one boggy mass with the interspaces filled with fibrin, while the serous exudate forms in very small amount, or is promptly absorbed as fast as it forms. In the intestinal walls and between them there will be found a small quantity of granulomatous tissue made up of tissue cells,

giant cells, and a few bacilli. The latter are seen often in the marginal portions of the giant cells. In this form ulceration of the intestine may take place, resulting in penetration of the walls at points of contact, and permitting the information of bimucous fistulas. In other instances localized abscess may form and finally open on the surface of the abdomen with a permanent fecal fistula.

In the suppurative form considerable encysted accumulations of pus are formed either by mixed infection from the bowel or from the breaking down of masses of tuberculous material. These types are always more severe than the ascitic form and less amenable to surgical treatment.

Treatment.—The treatment of tuberculous peritonitis must be varied according to the conditions presenting. In the early stages, by proper hygienic measures with fresh air, good food and sunlight, much benefit may be obtained especially in children. It is probably also true that the proper use of light treatment may prove beneficial. If response to these measures is not prompt, and particularly in the ascitic form, abdominal section is indicated.

The operation may consist simply of opening the abdomen, exposing the peritoneum to the air with a view of promoting an active reparative process.

An important question for consideration in this connection is whether the procedure shall end at this point or whether it shall include removal of an infected appendix or tuberculous tubes and ovaries. Undoubtedly when the latter method can be carried out safely it is good practice because the possibility of reinfection from these sources is removed, permitting and hastening a permanent cure in case satisfactory repair of the peritoneal involvement is obtained.

It is extremely doubtful whether the instillation of any chemical substance into the abdomen is productive of any particular benefit. Our experience makes us strong opponents of such treatment. Neither the use of iodoform or bichlorid of mercury, as employed twenty-five years ago, nor the more recent use of iodine or of sulphuric ether seems to be of any especial advantage in promoting a cure.

In the fibrinoplastic form the indication most frequently calling for operative intervention is intestinal obstruction. In some instances where the lesion is circumscribed, resection of the entire localized loop of intestine may be productive of a permanent cure. In such a case, if the condition of the patient justifies it, excision of the causative tuberculous adnexa or appendix may be undertaken, or this step may be deferred for a later date. Enterostomy may prove a life-saving

measure in this type of the disease. Very little can be done in an operative way with the diseased lymph-nodes in the mesentery, unless a limited number of the infected nodes lie in close relation to the diseased appendix or the involved localized loop of intestine. Subsequent to operation these individuals should have every aid possible from climate, sunshine, fresh air, diet, properly regulated rest and exercise, and such therapeutic measures as may be indicated. While light therapy is not yet fully developed, it appears to offer something. The same is true of Roentgen irradiation. But upon the intravenous medication, particularly of arsenic and iron, in conjunction with the measures mentioned above, must be placed the greatest reliance.

POSTOPERATIVE PERITONITIS

In the early days of abdominal surgery and somewhat infrequently at the present time clean abdominal operations are followed by a severe and occasionally fatal peritonitis. The careful attention to technical detail during such an operative procedure employed at the present time has greatly diminished the frequency of this occurrence. When such an accident happens in a hospital at the present day an explanation is expected both to determine its cause and to avoid future wound contamination as well.

A considerable percentage of such cases results from accidental soiling from the escape of the contents of a hollow viscus with contamination of the peritoneal surfaces. For such material to cause peritonitis it must contain a virulent type of organism, since it has been clearly shown that the peritoneum has a considerable resistance to ordinary pus-producing bacteria. The organisms most likely to be found as causing this type of peritonitis are the *Streptococcus viridans* and *hæmolyticus*, either in pure or mixed strain. Such contamination upon a damaged or traumatized peritoneum, especially if a considerable quantity of bacteria enter, may result in a violent infection of the type seen in septic peritonitis. Milder forms may assume a severe yet less acute type. The smaller contaminations result in localized peritonitis with tendency to result in either complete or partial ileus. The development of this type of peritonitis after abdominal section adds very materially to the gravity of the case.

Peritonitis developing after an abdominal section is, as a rule, promptly determined, but sometimes the symptoms are most confusing. Cases in which a mild infection persists along a vaginal stump or about an appendix or a sutured intestine where a small leak has occurred

just sufficient to cause an adhesion of the intestine and partial angulation, may confuse sufficiently to make unnecessary and often fatal delay. The occurrence of peritonitis is usually determined under these circumstances by the absence of visible peristalsis and by the absence of sounds under auscultation. The presence of persistent vomiting is important. Notwithstanding the great risk of a secondary operation I am impressed with the fact that when these symptoms arise, particularly with distention and inability to relieve the flatus, intervention is indicated. When the patient's condition does not justify a radical procedure, the work may be done under local anesthesia, even if nothing more than enterostomy is done.

Early opening in cases of less severity in which the primary operation has left the patient sufficient reserve is positively indicated. Rare judgment in the handling of such cases is necessary to a successful result. No hard and fast rules may be laid down for the conduct of such a case.

Puerperal cases always show the first evidences about the pelvis, hence are not as a rule so difficult of recognition. It is well to remember that certain other causative factors producing peritonitis may occur during the puerperium and must not be overlooked.

INTRAVENOUS ANTISEPSIS

The subject of blood stream infection is one of the greatest importance. This is particularly true at this time since the studies in pathologic physiology and of intravascular therapeutics have assumed considerable proportions.

Deaths from peritoneal infections frequently result not alone from the local process but particularly from the sepsis which accompanies the local lesions.

Septic intoxication is supposed to result from the entrance into the blood of certain chemical products of microbic growth, presumably ptomains. This has been considered the least dangerous of the forms of sepsis.

Septic infection has for a long time been recognized as the result of the presence of septic bacteria and their poison in the blood stream and their further proliferation there.

Pyemia results from the presence in the blood stream of pyogenic bacteria in colonies or clumps, which tend to result in embolism, infarction, and the formation of new abscesses in distant locations.

The difference between septicemic and pyemic infection lies in the

tendency to clumping of bacteria in the latter and its absence in the former.

For years the injection of drugs into the blood stream was thought to be a very serious proceeding. Recent studies in this direction following the work of Ehrlich have demonstrated that many substances may be safely placed within the blood stream with proper precautions. Some of these experiments have been sufficiently encouraging to foster the hope of ultimate success in overcoming blood stream infections.

The presence of bacterial contamination of the blood need no longer be surmised. By proper culture methods the presence or absence of pathogenic organisms can be positively determined.

In order that any use of intravascular bactericidal agents may be logically employed the culture of the blood should be positive. The use of bactericidal agents within the blood stream is but a step beyond the work of Wright, who sought to overcome infections by increasing the opsonins in the blood which would tend to combat the bacteria. The present method substitutes a direct attack against the enemy for a flank attack. There will be still something necessary to perfect methods of this type, even if bactericidal agents can successfully destroy the bacterial flora. The poisonous products are yet to be disposed of and the effect of massive destruction of bacteria upon the blood stream is to be considered. Even when the blood culture after the use of a bactericidal agent remains negative some of these patients go on to a fatality, probably because the blood is unable to assimilate the dead bacteria and the chemical products accompanying the infection. It may finally be shown that venesection may be a valuable adjuvant to such methods. The tentative study of graduated dosage will be very advantageous.

In 1919 H. H. Young, Edwin C. White, and E. O. Swartz reported upon a new germicide used in the genito-urinary tract, mercurochrome,¹⁷ which they found to be nontoxic, nonirritating, and very penetrating for use as a local urinary bactericide. Acting with Young, White made some 250 mercurials, some of entirely novel composition. Three of these were found to be very useful: mercurochrome, meroxyl, and flumerin.

Of these mercurochrome was found because of its penetrating property to be a most useful antiseptic. Laboratory studies by Miss Justina H. Hill at the Brady Institute have shown mercurochrome to be of value when given intravenously, not only as a urinary antiseptic, but also in general infections.²⁰

One of these cases diagnosed as colon bacillus pyelitis was sterilized

by one intravenous injection of 40 c.c. of a one per cent solution of mercurochrome, a dosage of 5 Mgms. per kilo of body weight.

Young calls attention to the interesting fact that in this case dead bacilli were found in the urine for three days, and after that no bacilli were found either on slides or cultures.

The work of Piper and the subsequent reports of Young seem to show that in mercurochrome and in gentian violet have been found a method of combating the severe blood stream infections which accompany so many general surgical infections.

In peritonitis blood stream infection is a very frequent occurrence, particularly in the puerperal form. Certainly there is sufficient evidence of the value of this new method of treatment to justify its employment in these otherwise grave cases.

In Young's report to the Southern Surgical Association December 11, 1923, are recorded seven cases treated by mercurochrome and five by gentian violet. Two of the cases treated by mercurochrome were of septicemia, both desperate cases, in which a cure was effected and the blood sterilized by intravenous injections of mercurochrome. The results were miraculous. The mercurochrome solutions seem to act efficaciously upon streptococcus septicemias, as well as those due to staphylococcus and colon bacillus.

February 18, 1924, since presenting the previous report, October, 1923, several septicemias have been treated, two *S. hemolyticus* and two *S. viridans* without success. On the other hand three cases of staphylococcus septicemia have been cured by the use of gentian violet injections.²¹

A few reports of salivation occurring from the use of mercurochrome injections have appeared. Apparently this does not seem to be of great importance although the dosage may be limited on account of it.

Some investigations have been made with other substances than the dyes mentioned by Young.

Beattacharje,³ a civil medical officer of Manipur, reports in 1919 the employment of iodine intravenously (0.2 in 8.0 c.c.), 3 minims being given with successful results in the treatment of severe infections from wounds produced by leopards.

The following year Gupta⁶ reported two leopard victims in a very septic condition who recovered from a similar treatment, 5 minims in one c.c. of normal saline solution being given.

DeCastro⁴ also reports success from the use of iodine in this way. Other substances have been injected, notably isotonic solution of sugar, Audian and Masmonteil,² peptone by Leclerc,¹⁰ according to Nolf's

method, 1.5 Gm. of pure peptone being dissolved in 30 c.c. of sterile water.

Perez¹⁴ employs 1 per 1000 of mercuric chlorid, giving 2 c.c. and rapidly increasing the dosage until 10 c.c. are given on the fifth day, and claims pronounced curative effect in septicemia.

S. Mello¹³ gave, as a last resort in 3 cases of puerperal typhoid or other fever, 2 to 3 Mgms. of mercuric chlorid by the vein twice in one day, then for two days 4 Gms. twice, and apparently moribund patients recovered.

Sulphate of magnesium after sterilization was used intravenously by J. A. Harrar⁷ in 2 per cent solution; 400 c.c. was injected in twenty minutes.

According to Dutton⁵ formaldehyd, 250 c.c. of a 1:5,000 solution intravenously, is a potent remedy in puerperal septicemia.

The injection of certain sera into the blood seems to rest upon an unsound basis. Recently the injection of solutions of radio lead have been made in the treatment of malignant disease with varying results. The present status of these researches is as yet uncertain and until further proof is given must be considered as experimental.

SUBACUTE PERITONITIS

Subacute peritonitis is the result of the acute localized form of this affection, or follows localization of a general peritoneal inflammation.

The tissue changes are similar to those occurring in the acute type of the disease. In this form there is always present a noticeable amount of reparative effort. This is evidenced by the presence of large numbers of adhesions. Abscesses of small size may be isolated by omental adhesions or agglutination of adjacent intestinal loops.

As long as the bacterial flora is capable of showing an active growth there will occur here and there an extension of the inflammatory process. In the majority of cases which progress from the acute to the quiescent stage there is present an increasing tendency to repair. With subsidence of the activity of the infection the reparative process goes on apace. Finally after a more or less protracted period the new formed adhesions are absorbed and the part is restored to normal. Because of the presence of a small number of organisms which are yet viable, localized activity may be observed. Under those circumstances the inflammatory exudate persists for a longer time. Small remnants, as bands or adhesions, remain permanently.

Symptoms.—The symptoms of this form of peritonitis are sim-

ilar to those of the acute variety. The most noticeable difference is in the lessened sharpness of the symptoms. The pain is dull and persistent. The distention is less marked. Vomiting is infrequent. Occasionally it is persistent. Much discomfort occurs from the presence of gas. Constipation is present. Appetite may be fair. Sometimes it is good. The temperature persistently ranges above normal. Leukocytosis is present and varies but little from day to day.

The diagnosis of this variety of peritonitis is made by the pain, the abdominal rigidity, the presence of palpable masses in certain regions, usually in the lower portion. In some instances these masses are palpable in the epigastrium. Occasionally the symptoms of ileus are present.

The prognosis depends upon the causative factor, the activity of the process and the resistance of the individual. It is not so grave as the acute type.

Treatment.—The treatment is conducted along general surgical lines. Expectant methods are best until the stage of quiescence appears. Some days are permitted to elapse after the febrile reaction has subsided before a section is performed. The probable outcome of such procedure must be carefully weighed particularly when the patient's condition is improving. More judgment is necessary in handling cases which seem to be improving, but for some reason begin to grow worse.

Many such cases demand operative intervention. Some will improve when food is withheld and may be carried to a safer time for operation. Almost all of these cases demand surgical measures before a final recovery is obtained.

CHRONIC PERITONITIS

The chronic variety of peritonitis is in the large majority of instances tuberculous in character. A few cases of chronic pelvic peritonitis appear as the result of gonococcic or pneumococcic infection, but these are better included in the group of subacute cases.

Actinomycosis.—Outside of the tuberculous form the rare type of actinomycosis must be mentioned. This type results from the presence of ray fungus, streptothrix actinomyces. It is usually secondary to actinomycosis of the intestine or pleura, most frequently the former. The invasion most often occurs in the lower ileum or near the cecum. This is a very rare lesion in this country.

The tissue changes are similar to those occurring in other portions of the body. The intestine and the peritoneum show evidence of in-

flammatory thickening. Isolated granulomata form, which tend to coalesce and to break down, forming a number of minute abscesses. These abscesses show a peculiar yellow pus, and carry the fungus which may be observed under the microscope.

The symptoms are moderately acute at times, but usually are slow in development. The process may be localized for some time. The part becomes brawny, indurated, tender, and painful. The stiffness and induration of the abdominal wall is characteristic, differing from the usual inflammatory rigidity. When the abscess opens on the surface the diagnosis may be made by the yellow granules containing the fungus. Many cases are not correctly diagnosed prior to operation, but the affection is so rare that it is not suspected.

The prognosis is not good. Some cases respond promptly to medication.

Iodid of potassium has been the standard treatment for many years. Some cases improve under its use. It should be given in rapidly increasing dosage to the point of tolerance. Some favorable reports have followed the use of arsenic.

The more serious complications of peritonitis are pneumonia, parotitis, pleurisy, meningitis, blood stream infection.

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CHAPTER VII

PNEUMONIA

Several factors enter into the development of pneumonia as an accompaniment of peritonitis.

A very important factor is the type of bacterial flora producing the inflammation of the peritoneum. Streptococcic and pneumococcic types are particularly prone to produce pneumonia. Pleurisy and meningitis also are likely to follow. These organisms seem to have a special selective action for the peritoneum, the lung, the pleura, and the meninges. Of these complications undoubtedly pneumonia is the most frequent.

The other factors entering into the development of pneumonia are the presence of a blood stream infection at the onset of the peritonitis. This occurs occasionally.

The necessity for anesthesia in the operative treatment adds another chance for pulmonary inflammation. The patient may develop an ether pneumonia. This occurs not directly from the ether, but because the evaporation of the ether chills the epithelium of the small bronchi, lessens their ability to empty the air cells of mucus, both because of the chilling and the anesthesia. The bacteria present in the air passages become active and temporarily the local resistance is lost. Therefore, pneumonia develops. Aspiration of vomitus or mucus favors the development of this complication.

Pneumonia may also occur as the result of embolism and infarction during the convalescence from acute general peritonitis. The development of sharp localized pain in the chest during an attack of peritonitis or shortly after operation for its relief is strong presumptive evidence of pneumonia. Rusty sputum or thick tenacious purulent expectoration is corroborative evidence. Dulness over a localized area with crepitant râles proves conclusively the presence of pneumonia. The x-ray findings are confirmative.

PAROTITIS

Occasionally in cases of peritonitis when the local condition seems to be subsiding the patient notices a swelling in the region of the parotid gland. Either one or both glands may be affected. The swelling

is accompanied by pain, tenderness, dryness of the mouth, and a foul breath.

In some instances the causative bacteria appear to reach the gland from the blood. In others parotitis appears to result from infection of the duct with bacteria within the buccal cavity. The presence of bacteria excites an inflammation of the secretory portion of the gland and the ducts as well. The secretion becomes diminished in amount and its character is changed as the result of the inflammatory process. The ducts become swollen, choked with bacteria and with thick mucus. Considerable swelling of the gland occurs and the process may continue to suppuration. Careless methods of anesthesia may be important in the production of this complication.

FISTULA AND SINUS

These conditions may follow an acute infection with abscess, traumatism, gunshot or stab wound, rupture of the intestine, rupture of the bladder, or where the suppurative process has continued until the abscess opened spontaneously on the surface. Fistula has also resulted from the infiltration of a neoplasm through the wall of the intestine and also through the abdominal wall.

In one of the author's cases the patient presented with a discharging stercoral fistula, connected with the ascending colon, the original abscess following perforation of a carcinoma having previously been incised.

In certain cases of intestinal tuberculosis, this condition may develop. More rarely fistula may follow spontaneous recovery from a strangulated hernia. Stercoral fistula is occasionally met in connection with a foreign body in the peritoneal cavity. More frequently a persistent suppurative sinus is met from this cause. I have personally seen a four-way stercoral fistula with two communications into the intestine and two into the bladder. Brief mention of such a case seems worth while, because in addition to the persistence of the fistulas which had resulted from the opening of an abscess of the abdominal wall prior to the patient's entrance into the hospital, there developed during his convalescence from the operation done for its relief an ileus which seemed complete and likely to prove fatal. His condition was looked upon as necessarily fatal. Fortunately, under rest and gastric lavage, he was tided over and finally made a recovery. In all probability the obstruction was due to kinking, because of gaseous distention, and after lavage of the stomach this was relieved. It is probably true also that a dose of

morphia which had been given to produce rest lessened the peristalsis and this lessened the angulation.

Another peculiar feature of this case was that the onset of this attack of ileus was evidenced by a typical epileptic seizure, with the prodromal cry, spasmodic contraction of the arms and legs, a brief period of unconsciousness, frothing at the mouth, and biting of the tongue. There was no history of epilepsy, nor were there any epileptic seizures afterward.

This case occurred during the period when some surgeons were recommending colectomy and sidetracking of the colon for epilepsy, and for autotoxemia. Considerable speculation occurred as to the causative factor for the epileptiform seizure. This form of seizure is often seen in infants suffering from intestinal parasites or from other alimentary irritations. It is not believed that this case even in part proves the contention of some that epilepsy is due to a stasis of feces in the colon.

Many cases of stercoral fistula and of persistent suppurative sinus follow surgical procedures for the relief of suppurative or other intra-abdominal lesions. In the early work in abdominal surgery, when silk ligatures were much in vogue, and where drains, especially rubber tubes, were almost universally employed, suppurative sinuses were frequent and fecal fistulas were by no means rare.

The planting of a silk ligature about the pedicle of a suppurative lesion of the fallopian tubes was often followed by sufficient contamination to prevent the peritoneum from being able to surround the foreign substance and make it innocuous. The usual result was that while the patient recovered from the illness and from the operation, the wound sometimes healing *per primum*, there appeared, either in the wound or in the cicatrix, an indurated spot which became painful and inflamed, finally coming to suppuration. This small abscess either opened spontaneously or was punctured with a knife. After the pus escaped the abscess failed to heal, and a few drops of pus appeared daily on the dressing. This condition continued often for considerable periods of time. Recovery finally took place after the extrusion of one or more knotted loops of silk.

In cases where von Mikulicz tampons were inserted into the abdomen or pelvis to wall off the localized suppurating area from the general peritoneal cavity, or where smaller gauze wicks were employed for drainage, it was not unusual to observe a fecal flow upon removal of the gauze.

The same thing sometimes, though less frequently, followed the

employment of glass or even rubber drains as the result of pressure upon a previously damaged intestinal wall.

Very often in the old cases of "green groin" from delayed operation in appendicitis, the surgeon was often glad to save the patient, even with a fecal fistula.

It also occurred occasionally in other lesions in which the dissection and enucleation were difficult and the wall of the intestine too friable to hold any sort of suture.

Both of these conditions occur less frequently at the present time, due to improvement in technical methods. The abandonment of silk pedicle ligatures and the adoption of catgut has practically done away with the sinus from this source.

Time is an important factor in the treatment of both sinus and fistula. If there is reason to believe that no foreign body has been left in the abdominal cavity, and if the patient is not losing ground, the expectant treatment may be carried out for some weeks, before any radical treatment need be instituted. Of course, if a foreign body is known or strongly suspected of being left, there is no question but that intervention is indicated, provided there is probability that the patient will stand the operation.

There are a few points of difference in the management of sinus and fistula, hence each will be considered separately.

Treatment of Suppurative Sinus.—This should be as simple as possible, and some weeks should be given for the foreign body, silk or catgut ligature to come away. We have found it bad practice to pack such sinuses or to do much probing. If the external opening is fairly free and the granulating walls of the sinus fairly strong, gentle installation of peroxid of hydrogen will in some instances loosen and aid in the evacuation of the suture. It should not be used where its exit is not free. Failing the extrusion of the ligature, after several weeks, if the patient's condition justifies it, operation may be performed to excise the sinus and remove the foreign body.

Treatment of Stercoral Fistulae.—Most cases of fecal fistula from appendicular abscess, from pelvic abscess, or following injury or resection of the large intestine heal spontaneously, provided the opening in the intestine is not in direct contact with the skin margin of the wound, permitting the mucosa to unite directly with the epithelial surface of the skin. Such a fistula does not heal spontaneously. Where, however, there is a granulating surface or canal communicating the lumen of the gut with the cutaneous surface, healing is likely to occur. Where there is more than one opening into the intestine, or where the rent is

large, this fortunate result may not obtain. The higher the fistulous opening is situated in the intestinal canal the less the likelihood of spontaneous healing. The reason for this lies in the fact that the intestinal secretion is poured out freely in the upper portions of the tract. It is very irritating to the skin and to the new granulations; hence it interferes with prompt healing. Another and perhaps more important factor is the fact that the rapid escape of intestinal juices and of food products interferes very materially with the nutrition of the individual, so that he may die of exhaustion in a short time. Wound healing occurs but slowly in badly nourished individuals. Elective enterostomy as a matter of treatment may be done with much greater safety in the distal portions of the intestine.

In the treatment of stercoral fistula it is always well to give a fair amount of time so that a protective healing around the fistula can occur.

If the lesion is in the lower ileum or large bowel and the patient nourishing reasonably well, it is safe to wait indefinitely, and in many cases this patient waiting will be well rewarded. While such expectant treatment is employed measures must be taken to protect the surrounding skin from the irritating discharges. Nothing is so satisfactory as cleansing the skin with 60 per cent alcohol, followed by the application of zinc oxid ointment. This should be changed daily at least. The dressings should be changed as often as they are soiled. By this plan of local treatment these patients are made fairly comfortable.

It has been found to be bad practice to insert any sort of drain into a fistula. Probing is unnecessary and likely to do harm. An occasional dose of castor oil prevents overloading the intestine, and occasionally a rectal injection of plain sterile water may be indicated. However, a fair degree of constipation is permissible, since the opening in the intestine will close best when only a small amount of feces passes through it, but overdistension of the intestine should not be permitted to occur.

These complications generally occur in patients who are extremely ill and who may not be able to withstand a second abdominal section until considerable time has elapsed. This gives the tissues a chance to correct the complication while the patient regains his vigor. During this period the diet must be most nutritious and carefully directed by the attendant. In cases where the lesion is high in the jejunum and where nutrition is not carried on properly, surgical measures must be employed even in grave cases, the lesser of two evils being accepted.

When the expectant treatment has failed and where, for reasons of

nutrition, operation becomes imperative, the surgeon must decide what is the best and safest method to bring about a cure. Many different types of operation have been proposed to correct this condition. The use of clamps bringing pressure on two adjacent loops has been recommended because it can be performed without anesthesia or under local anesthetic. Various suture methods have also been used with varying success. Most of them are unsatisfactory and uncertain. In cases where the external opening is small, where a limited amount of intestinal contents escape and where the mucous membrane has not lined the fistula, the actual cautery inserted into the fistula may bring about healing. This may be attempted with perfect safety followed by a compress snugly held in place. Failing by this method to get prompt healing and where there is reason to believe healing will not take place, also where a foreign body is lying at the interior extremity of the fistula a radical abdominal section is required. The same sort of operation is indicated in the case of a persisting suppurating sinus.

Any patient whose resistance is good should be able to withstand the necessary steps to obtain a cure of these annoying complications. The usual preparation for section is made, and the outlet of the fistula cauterized, either with actual cautery or carbolic acid. In some cases a gauze wick is placed in the opening and a ligature or forceps placed about it. In the operation itself a certain amount of care is to be employed. It has been found advisable in making the incision for the treatment of a sinus or a fistula to open the abdominal wall at a point some distance away from the cicatrix in which the fistula is situated. In the upper abdomen the new incision is begun below the scar, in the hypogastrium above the scar. This should permit the passage of two fingers within the cavity to encircle the fistula and to see that no intestine lies in contact with the wall or fistula at this point. Then the old cicatrix including the fistula is excised. The attached omentum and intestinal coils, after gauze protection is placed about the field, are separated from the fistulous tract, by careful dissection. The internal ostium of the fistula is soon found. In some instances there is such agglutination of the intestinal coils that a resection of the loop is the quickest way to deal with it. Usually, however, the tract may be readily traced to the intestine, isolated and excised. The opening in the intestine should be sutured in a transverse manner to the direction of the gut so as to prevent angulation. All raw surfaces are to be sutured to prevent adhesions and the abdominal wound closed without drainage. This method may be made very simple in competent hands, but is always a serious undertaking for the inexperienced or timid

surgeon. When properly performed with careful methods of suturing, the results are brilliant.

Following the operation the patient is placed in bed and when conscious he assumes the Fowler position and takes plenty of water by mouth. Proctoclysis may be employed, but it is best to wait twelve or twenty-four hours and give one half pint at a time, particularly if the colon is involved. In case of marked dehydration, subcutaneous infusion of normal saline is beneficial. Let the patient alone, so that healing may take place. The administration of morphin is advisable, if necessary, to keep him quiet. One fourth grain usually is sufficient.

Have no worry about a bowel movement even for four or five days. Usually that takes care of itself. Plenty of water by mouth and early feeding are indicated.

Of all the methods proposed for the relief of these complications the one described above has been most satisfactory in the writer's hands.

POSTOPERATIVE VENTRAL HERNIA

This type of hernia follows abdominal section in a small percentage of cases.

Masson reports during a period of four years 28,970 abdominal operations performed at the Mayo Clinic. Of these 596 or 2.05 per cent were for the repair of postoperative hernia.

During this same period, 4,249 inguinal, 217 femoral, 327 umbilical, and 113 miscellaneous hernias were repaired. The postoperative hernia constituted 14.66 per cent of the total operations for hernia. In 134 cases the primary operation was performed at the Mayo Clinic and 462 elsewhere.

Postoperative hernia occurs oftener in hospitals handling acute abdominal conditions or emergency operations. This result obtains because of the greater frequency of infected cases coming into such hospitals. The percentage of hernia developing in clean cases is very small.

The most important causative factors in the production of this type of hernia are suppuration and the use of drainage. The presence of peritonitis of sufficient degree to produce temporary adynamic ileus with its coincident intestinal distension with the nausea, vomiting, and hiccough tends to result in weakening of the line of suture.

Occasionally a small piece of omentum is forced between the sutures by the great intra-abdominal pressure. Subsequently such a defect is enlarged by intra-abdominal pressure straining, coughing, etc., until a

considerable hernia is established. Rarely the entire wound may give way and permit the intestine to escape.

The injury of the nerve supplying the abdominal wall is an important factor in the production of hernia and the lack of tone in the aponeurosis makes more difficult the successful repair. Foreign bodies accidentally left in the wound also tend to result in hernia. Likewise fistula and persistent sinus has the same tendency.

Imperfect suture methods, poor suture material, imperfect control of hemorrhage all tend to favor the formation of postoperative hernia. Such occurrence should always be in the mind of the operator in closing an abdominal wound. Painstaking care in abdominal closure is as important as in any portion of the operative technic.

The avoidance of the occurrence of hernia after abdominal section can only be accomplished by care in every step of the procedure. In prevention of the occurrence of this sequel the placing of the incision is important. Median line incisions are preferable when the surgical difficulties are not increased to the embarrassment of the operator. This is because the damage to nerves and musculature is less than in other portions of the wall. It is very important to preserve nervous structure. Control of hemorrhage, even slight oozing, is important since blood clot between the wound edges favors infection, delays healing, and results in a weak wall. Each tissue is coapted with the corresponding structure and dead spaces avoided. Suture material which is sterile and sufficiently durable to last the expected time is necessary. Stitches applied too tightly result in sloughing and weakness of the wall. After the suturing is complete the dressings are held in place by adhesive support.

Every possible measure for prevention of pulmonary complications is to be employed since these affections lead to coughing, which develops weakness in the healing wound. These consist of careful oral asepsis, skillful methods of anesthesia, the avoidance of postoperative nausea and vomiting by gastric lavage. After the operation simple measures to prevent and overcome abdominal distension are employed.

Operative Treatment.—The same careful technic should be employed in these cases. The lines of incision should be made so that the approach to the peritoneum will avoid adhesions and injury to the viscera. Scar tissue which will prevent prompt repair may be excised. The peritoneal structure lining the pockets of the sac is dissected off. The different layers of the abdominal wall are isolated and coapted, the peritoneal layer as well as the aponeurosis being carefully sutured.

Subsequent to operation these patients should refrain from heavy work for some weeks until the cicatrix has become firm.

CHAPTER VIII

CONDITIONS CONTRIBUTING TO PERITONITIS

DIVERTICULITIS

This occurs in two distinct affections. One affects Meckel's diverticulum; the other affects the diverticula usually occurring along the colon, rarely in the small intestine.

The first form, or Meckel's diverticulitis, assumes an acute type similar, except as to location, to an appendicitis. This appendage of the gut, the remains of the omphalomesenteric duct, may become acutely inflamed. Adhesions to other structures may form as the result of such inflammation. There is no separate blood supply to this structure, which receives its nourishment from the blood-vessels of the intestinal loop at its base. Marked inflammation in this loop may be accompanied by inflammation in Meckel's diverticulum. Enteroliths may form within it and by ulceration produce a localized peritonitis and abscess around this rudimentary structure.

The symptoms of this affection closely resemble those of appendicitis with the exception of the location of the pain, rigidity, and tenderness, which are frequently left-sided. Some cases are mistaken for appendicitis, an error which is unimportant since the management of both conditions is the same.

Acquired Diverticulitis.—Inflammation of acquired diverticula of the colon according to McGrath was first associated with isolated circumscribed adhesive peritonitis by Graser in 1899.

Almost all of these diverticula according to McGrath are of the false type. They consist of a herniation of the mucosa through the muscularis, usually at the site at which the latter is penetrated by vessels. They are most common in the appendices epiploicæ and occur in the sigmoid. A few cases have been observed connected with the small intestine. Charles M. Watson mentions a case of multiple diverticulitis occurring in connection with the jejunum; occasionally they are present in the duodenum.

These diverticula vary considerably in size, some reaching the size of a hen's egg. They are rounded or oval sacs with constricted openings into the intestine. Usually they contain firm fecal material or fecaliths.

They are seen most often in adults of sedentary habits, who have long been subject to constipation. Often they exist for long periods without producing any inflammatory changes or evidences of any other abnormality.

Histologically the sac wall is formed of mucosa, submucosa, and serosa. Very little muscular tissue is found in the wall. The serous covering is often very loosely attached.

The presence of feces within the sac without occasional evacuation into the intestine will eventually result in irritation. The latter may continue to ulceration and infection of the walls of the sac and adjacent tissue. This process is of necessity slow, the irritation being kept up because of the persistence of the foreign body which can with difficulty escape.

There is a chronic form of inflammation accompanied by round cell infiltration of the wall of the intestine and adjacent structures. There may be so much of this indurated tissue that it resembles carcinomatous infiltration and may be mistaken for it.

Whether such inflammation has any more direct relation to the development of carcinoma of the colon is not yet determined. A marked spastic contraction of the intestine is seen in some cases. Louis B. Wilson mentions 4 of a series of 15 cases which showed malignancy. Some of these inflammatory processes continue to abscess formation. In 5 of W. J. Mayo's cases intestinal obstruction resulted.

Symptoms.—The symptoms of diverticulitis are slow of development. This affection occurs most often in individuals who are inclined to obesity, but thin persons are not immune.

A sallow complexion is often observed, but there is no cancerous cachexia. Loss of flesh is not a prominent symptom. The chief complaint is soreness, tenderness, weight, and discomfort in the left lower quadrant. A palpable tender mass is sometimes observed. Constipation has been present for long periods and occasional attacks of temporary diarrhea may alternate with it. Blood is not present in the stools.

Proctoscopy rarely detects any evidence of the condition unless the diverticulum is situated very low.

Diagnosis.—The diagnosis may be tentatively made from the history, the presence of a tender mass over the sigmoid or descending colon. The roentgenologic examination will usually give confirmative evidence and is the most valuable means of diagnosis at hand.

Treatment.—Localized diverticulitis may be treated by direct excision. Localized abscess arising from this source calls for free drainage. Some of these cases will require subsequent surgical procedure.

Cases producing obstruction or marked hyperplasia of the intestinal wall are best treated by resection of the intestine.

RELATIONSHIP OF APPENDICITIS TO PERITONITIS

No discussion of the subject of peritonitis would be complete without some consideration of appendicitis as a causative factor.

This affection about which there was so much confusion before the appearance of the classical discussion of the subject by Reginald Fitz, in 1888, is probably the most frequent single cause of peritonitis.

In the old days patients with appendicitis were considered to be suffering from typhlitis or from perityphlitis. A large percentage of such patients died from inflammation of the bowels, so-called. Since that time, largely due to the work of American surgeons, the subject of appendicitis has become familiar to the profession throughout the world and the lives of very many individuals have been saved by surgical intervention.

The relationship of inflammation of the vermiform appendix to peritonitis is very close. In the acute type of appendicitis when the active bacterial agent infiltrates the wall of the appendix, choking both the lymph and the blood-vessels, there follows in a short time a reaction of the adjacent peritoneum to such infiltration. In the mildest of the acute forms this reaction is slight. Not infrequently the omentum grasps the appendix as it were and surrounds it just as a finger may be grasped by a hand. The peritoneal exudates under these conditions seal the appendix completely away from the general peritoneal cavity and protect it from a general contamination. In the same mild type, seen often in tuberculous appendicitis, as well as in pyogenic infections, the process may terminate at this point. The contents of the distended appendix may discharge into the bowel and the vessels become freed of the crowding bacteria and the part return to normal. The peritoneal inflammation subsides and when the adhesions formed in repair have not existed for too long a time they are in many cases entirely removed by absorption.

Where the process only partially clears up and the appendicitis slumbers along, the adhesions become more and more firm. In some instances from this chronic state of inflammation the appendix undergoes obliteration, perhaps from the tip toward the base. In rarer instances its lumen is obliterated at some point near the middle or even at the base. This condition may lead to the exhibition of symptoms sufficient to bring it to surgical operative treatment. Or there may occur, as a result of such occlusion, a distention of the lumen of the

appendix until it may be considered as a cyst. The contents of such a cyst are a glairy mucus, some detritus and perhaps an enterolith.

The more chronic the inflammatory process in appendicitis the more likely are adhesions to result, which, becoming more or less permanent, tend to result in chronic intestinal stasis, internal ileus and volvulus.

The more acute forms of appendicitis run a rapid course. Frequently a violent appendicitis, developing suddenly, becomes in a few hours a source of bacterial contamination of the entire peritoneal cavity. The inflammatory process spreads rapidly and may extend to every part of the peritoneal sac, without any attempt at delimitation. Such a course usually means a low resistance and the leukocyte count as a rule is low.



FIG. 39.—ADHESIONS BETWEEN OMENTUM AND INTESTINE IN APPENDICITIS.

The term fulminant has been applied to such cases. The appendix is "strutted," leaking, or gangrenous. The peritoneum is glazed at first and quite dry, becoming fiery red, swollen, and moist in a short time. Flocculent masses of lymph are seen here and there. A considerable amount of free fluid is present in the sac, either a yellowish tinged flocculent serum or a purulent material.

In cases of this type which become quiescent there are formed between adjacent coils, between these and the omentum and also between the omentum, the intestines, and the abdominal walls a mass of adhesions (Fig. 39). These are often productive of acute ileus and may result in intestinal stasis of a chronic type with acute exacerbations.

In another group of cases where the local process continues to rupture of the appendix with agglutination of the intestines and mesentery

over it, there forms an abscess. When such rupture of the appendix occurs in a walled-off space, notice comes to the individual by the sudden cessation of a previously severe pain. The abscess forms rapidly after such an occurrence, and in those cases in which peritoneal adhesions are sufficiently strong to retain the pus in this new sac the local condition remains quiescent for a time. During such a period the cavity of the abscess increases in size. Once more pain is evident with the other cardinal symptoms of appendicitis: vomiting, rigidity, tenderness, a febrile reaction, and a palpable mass in the right lower quadrant. When the tension in such an abscess becomes such that the adhesions can no longer protect against it, rupture occurs and there follows an acute general peritonitis. The onset of this inflammation is evidenced by severe collapse, followed by pain, vomiting, general abdominal rigidity, and tenderness. Both the latter symptoms are more prominent on the right side. The mass may disappear after rupture or may be still palpable. Transportation of a patient with a tense appendicular abscess is dangerous and should be conducted with care.

There can be no question at the present day whether acute appendicitis should come to operation at once after the diagnosis is made when the patient's condition will permit. This plan of treatment is the best known method of preventing peritonitis. When the fulminant or the perforative type is present and seen early, again a section is indicated immediately. The rare exception to this rule is marked depression and collapse, which clearly indicate that the patient can not withstand the additional tax of an operation. Such cases may be brought to an operable stage by the Fowler position, Murphy drip, abstinence from food or drink until the vomiting ceases. Gastric lavage as often as necessary is valuable. Ice to the abdomen will allay pain. These measures are useful in the prevention of peritonitis from this source and in its control when once developed.

Cases otherwise operable, when operation is refused, must be conducted along similar lines.

Next in importance to the development of acute peritonitis are the results of the adhesions arising therefrom. Chronic appendicitis will be considered here particularly in its relationship to the production of adhesions and as an incidental factor in acute attacks of the disease.

That the relation of a chronic engorgement or a chronic inflammation of the appendix to an acute attack is close, must be admitted. The contention of some authors that a large proportion of all acute inflammations of the appendix have been preceded by recurrent attacks of more or less chronic type does not hold true. In the majority of acute

cases there are no evidences of a previous inflammation either as shown by adhesions, by infiltration of the wall of the appendix or the presence of fecaliths.

It is more probably true that, following a mild acute attack or even a severe one, a chronic, persistent or recurrent inflammatory process remains. In such a case there may develop in the process of repair a partial constriction in the lumen. Under such conditions there is a tendency for fecal material to accumulate. These fecal masses reach a size which makes difficult their extrusion into the gut. Therefore, they remain within the appendix and act as irritants. These foreign bodies may cause congestion and even ulceration of the mucosa or the entire wall of the appendix, resulting in rupture.

Other cases show no evidence of cicatrization and stenosis, but the walls remain more or less engorged and the vessels in some portions are choked with bacteria. When such changes are present in the walls and in some cases during the original acute attack, adhesions are formed binding the appendix to adjacent organs, causing angulation of the appendix itself and, in some instances, of the intestine as well. These adhesions, in turn, are potential causes of peritonitis since they sometimes produce strangulation of the intestine.

So long, therefore, as an appendix shows any signs or symptoms of inflammation such as soreness, pain, tenderness, rigidity, or a palpable mass, it is a potential menace to the life of the individual. This state of chronic or recurrent inflammation in this organ tends to lower the resistance of the individual, to produce disorders of digestion and even distinct pathology in other structures. Phlebitis in the veins of the part has been noted.

Some authors, notably Moynihan, claim a distinct relation between appendicitis and gastric ulcer.

The adhesions about a chronically inflamed appendix are likely to become quite dense. Occasionally a very small abscess may lie for some time quiescent at the site of the appendix to explode later in a fulminant attack of the disease.

The extent of the adhesions depends largely upon the amount of local irritation produced by this type of inflammation. These, in turn, may cause partial stenosis, resulting in milder cases in the production of constipation or in chronic intestinal stasis or in other instances in acute mechanical ileus.

The presence of a diseased appendix of the chronic type may be made out by the history of one or more attacks of right iliac pain and tenderness, with slight tenderness to region of the appendix.

There will usually be increased leukocytosis, but this is not marked. The diagnosis in some cases may be facilitated by radiologic studies. The treatment consists in the prompt removal of the organ.

In certain types of the chronic cases there is a cicatrizing process which is obliterative in character. Considerable discussion has occurred as to whether this type of cicatricial obliteration is inflammatory or congenital in origin. It seems probable that both types occur.

The extent of the changes which have taken place in an appendix sufficient to place it in this group depends largely upon the observer. Careful observation should result in a correct recognition of the condition. Sufficient pathology should be found at operation to justify the diagnosis.

It is well to avoid placing all obscure intra-abdominal pain low on the right side in the group of appendicitis. To avoid the chagrin of operating in cases where no pathological changes can be found in the appendix, the greatest care in detailed history taking and physical examination is necessary. This followed by a close and careful physical examination will bring accurate diagnosis.

The conditions likely to be confused with appendicitis of the chronic type are renal or ureteral calculus, chronic inflammation of the gall-bladder. In the female chronic inflammations of the tube and ovary may prove misleading.

The presence of symptoms sufficient to justify a diagnosis of chronic appendicitis is sufficient ground for advising its removal as a preventive measure in the treatment of peritonitis. In all doubtful cases urinalyses and x-ray examination should be carried out to exclude stone.

The dyspepsias, the headaches, the renal, vesical, and pulmonary disturbances are not all due to this type of appendicitis nor will operation relieve the symptoms in every case. Before subjecting a patient to operation for chronic appendicitis the laboratory findings and the clinical evidences should point strongly to the correctness of the diagnosis.

Psoas Abscess.—In young individuals as a result of spondylitis of tuberculous origin, a psoas abscess develops. When such an abscess is situated on the right side it may present symptoms very similar to appendicitis. This similarity is due to the swelling in the right iliac region, the attitude of the limb, and perhaps a slight fever. Such symptoms are presented in the slower forming types of appendicular abscesses. It is important to make the diagnosis of the latter condition early because of the danger of the development of peritonitis.

Appendicitis is almost always accompanied by vomiting at its inception. The pain in appendicitis is situated near the umbilicus at

first, is subject to exacerbations and finally localized over McBurney's point. Tenderness and rigidity are prominent. Leukocytosis is present.

In psoas abscess vomiting is absent. The pain is never as sharp as in appendicitis, but is dull in character, being more of a soreness. The limb is held more firmly in the flexed position. On efforts to extend the thigh there is marked lordosis. In a young child it is easy to elicit evidence of rigidity of the spine, on lateral flexion. The patient having Pott's disease always holds his back rigid in stooping, but not so with appendicitis.

The cases most likely to be confusing are the very early cases of psoas abscess and the slow more chronic types of appendicitis. The skiagram should finally show evidence of disease of the vertebræ. Rarely will an aspirating needle be employed for diagnosis in this locality.

DISEASES OF THE KIDNEY IN RELATION TO PERITONITIS

It is remarkable how infrequently the kidney is responsible for peritoneal infections, notwithstanding the fact that it is very important in eliminating bacteria from the body.

The reason for the comparative immunity of the peritoneum to such infections probably lies in the resistance of the renal capsule proper, the intervening fibrous capsule and the resistance of the healthy peritoneum.

The conditions which may result in contamination of the peritoneum are suppurative kidney, purulent pyelitis, tuberculosis of the kidney, and hydatids. The actual frequency with which these affections implicate the peritoneum is slight and is only mentioned that their possible relationship may be kept in mind.

Hydatids involve the peritoneal structures with relative frequency, at least 4 in 18 cases being reported in which the intestines were closely matted together in hydatids of the kidney (Fig 38).

J. L. Kretschmer has made comprehensive report upon the subject of hydatids of the kidney, and gives the histories of eighteen cases occurring in the United States and Canada. The frequency of hydatids of the kidney is small, ranging from 0.21 per cent to 5.4 per cent in cases of echinococcosis.

Symptoms.—These patients complain of discomfort in the side, sometimes of acute pain. Occasionally small cysts may pass and these or hooklets be found in the urine.

Hematuria has been present in some cases. The kidney is often

enlarged and tender. Abdominal soreness and distention are sometimes observed. There is progressive loss of strength and flesh.

Nephrolithiasis.—This condition is indicated by renal colic with the presence of blood and pus in the urine. The blood may be in microscopic amount or may be visible to the naked eye. An attack of renal colic may confuse the diagnosis both during the early stages of peritonitis or its causative appendicitis, or during the late stages when adynamic ileus or intestinal paralysis is present in peritonitis.



FIG. 40.—ECHINOCOCCUS DISEASE OF THE KIDNEY. BISECTION OF KIDNEY.

(Redrawn from Kretschmer, *Surgery, Gynecology and Obstetrics*, February, 1923, 196-207.)

Often in attacks of renal colic the very severe pain may produce temporary paresis of the intestine. The abdomen sometimes becomes greatly distended and the patient is in evident distress. At this time perhaps the urinalysis has not been made, thus adding to the difficulty in diagnosis.

The important points in differentiation are the character of pain, the absence of fever, the presence of pus and blood in the urine in nephrolithiasis. The pain in renal stone is sharp, lancinating, appearing usually in the loin, and extending down the course of the ureter along

the groin and to the end of the penis. There is always pain on deep percussion in the loin by the method of Israel. Relief of such pain is as sudden as its onset. Vomiting may or may not be present.

The stoppage of fecal flow is only temporary in renal colic. There may be complete stoppage in peritonitis. A blood count usually shows increase in the number of leukocytes.

The history of the affection just preceding the peritonitis is of considerable aid in the determination. It is well to remember that a patient with appendicitis may also be suffering from renal or ureteral stone. Stone in the right ureter is particularly confusing. The tenderness is less diffuse. There is less rigidity, no fever, and usually blood or crystals in the urine. The blood-picture is normal.

With these facts in mind the diagnosis may be made. The presence of blood in the urine will make it wise to defer operation until a skiagram of the urinary tract can be made. When a stone is present, as shown by the shadow, an unnecessary appendectomy may be avoided.

Hydronephrosis.—Hydronephrosis which develops from intermittent obstruction of the ureter sometimes reaches considerable size and must be differentiated from cyst of the pancreas, mesenteric cysts and from retroperitoneal tumors.

Renal tumors such as hypernephroma and sarcoma are also to be distinguished from peritoneal lesions.

The fact that the ureter is obstructed may be determined upon catheterization of this duct. Roentgenograms taken after the insertion of a leaded catheter into the ureter, or the use of pyelography, will outline the kidney, its pelvis, and the ureteral tract, showing its relationship to the mass and making the diagnosis positive.

Perirenal Infection.—Perinephritic abscess may result from infections within the kidney or arise from without. The conditions most likely to show perirenal suppurative inflammations are renal stone, tuberculosis and acute suppurative infections (the so-called suppurative kidney).

It is remarkable in view of the many suppurative diseases of the kidney how seldom the process extends to the perirenal tissues. This is due for the most part to the strong and resisting capsule of the kidney. Prolonged and persistent pressure within the capsule, however, results finally in the softening of this structure so that contamination of the fatty tissues adjacent takes place. It is a well-known clinical and pathological fact that pressure from fluid or semisolid material against the firmer structures eventually causes the latter to give way.

Infection of the perirenal tissue also follows peritoneal or retroperi-

toneal infections in a large number of cases. This is particularly true of the right side because of the frequency of appendicitis and the occasional phlegmon of the retrocecal tissues which follows this lesion.

Perirenal infections of severe type are sometimes mistaken for peritonitis. The similarity rests on the right-sided pain, occasional nausea, and vomiting. If due to renal stone there may be a history of repeated attacks of pain which were thought to be due to appendicitis.

Cases of retrocecal appendicitis simulate rather closely the symptoms of perinephritic abscess and of nephrolithiasis as well. Both perirenal suppuration and appendicitis show an increase in the leukocyte count. Bimanual palpation will usually indicate the location of the swelling.

Rupture of gastric or duodenal ulcer, suppurative pancreatitis, abscess of the liver, may be followed by perirenal abscess. Such abscess rarely causes acute peritonitis.

Symptoms.—These suppurative processes usually are announced by a chill, sometimes severe, with a rather sudden rise of temperature, pain in the loin, abdominal distention and general tenderness. The tongue is coated. Constipation is frequently present. The urine is scanty, high colored, and may be suppressed. Leukocytosis is increased. In a short time a palpable mass may be felt in the region of the kidney, giving a suggestion of enlargement of that organ. Blood, pus, and occasionally crystals are found in the urine, yet these may be absent.

The picture of a perirenal suppuration is so characteristic that it should be made out definitely from the above symptoms. It may be confused with a postcecal abscess or right-sided localized, suppurative peritonitis following appendicitis. The latter, however, gives none of the urinary findings, and the mass is distinct from the kidney.

INTESTINAL ADHESIONS

This term is applied to the more or less abnormal and permanent attachment of two peritoneal surfaces. Under the term adhesions are included agglutinations, those temporary plastic formations which hold the abraded intestinal surfaces in contact immediately after an injury; those short close permanent structures which hold coils of intestine together without serious constriction of the lumen; adhesions, proper; and those somewhat elongated structures which cause traction upon the different viscera and frequently acting as a snare or by direct pressure cause obstruction, known as bands.

In contradistinction to congenital bands, adhesions as here described are always the result of reparative effort upon the part of the organism

to overcome the effect of traumatism, mechanical, chemical, or microbic in origin. Viewed from this standpoint these structures are by no means so baneful as they are usually considered. Without these plastic reparative processes the protection of the peritoneal tissues against traumatism of all kinds could not be obtained. As a matter of fact the reparative process in peritoneal tissues is one of the most active in the body.

Immediately following a perforation of the intestine the reparative process tends to seal the opening, thus preventing leakage. Again when the intestinal contents escape the same processes attempt with some degree of success to isolate and surround the noxious material, thus preventing its spread over wide areas.

These peritoneal reactions are utilized by surgeons in handling wounded viscera, also in protecting the peritoneum locally and the entire organism as well. The entire problem of drainage, including the cofferdam of von Mikulicz, is based upon a correct appreciation of these reparative processes. One of the most beneficent effects of adhesive formations lies in the ability of the peritoneal tissues to remove surplus material utilized in the process of repair. A distinction should be made between the temporary and permanent reparative tissue. Adhesions, therefore, may be classed as temporary and permanent.

Temporary adhesions always form in cases of trauma and in all types of inflammation of the peritoneum which persist a sufficient length of time, the exception being those cases in which death ends the scene from the virulence of the poison before such reparative forces become active, as in septic peritonitis. The process may be just sufficient to repair the damage, and when this is accomplished the part shortly becomes restored to normal. When the damage is excessive and when the infection persists, especially when certain particular organisms are the causative factor, the adhesive material continues to form and permanent adhesions result.

Serous surfaces whose endothelium is intact do not adhere. The endothelial cells of the peritoneum are placed in such a thin layer that even the most gentle manipulations may damage them or separate them from the limiting membrane beneath.

The effect of exposure of peritoneal tissues to the air for considerable periods of time is familiar to all surgeons. The surface becomes congested and somewhat less smooth from diminution of the normal secretion. Later a plastic coagulable material forms which tends to hold the surfaces together. When dry gauze is permitted to rest in contact with the serous surface these changes are aggravated. Moist gauze applied too hot produces similar results. Any foreign substance

coming into contact with the peritoneum excites similar changes. Chemicals act in the same way. Whenever the trauma is sufficient to denude the endothelial layer a serofibrinous exudation takes place, and this material has adhesive tendencies.

It is only after the vitality of the cells has been impaired that fibrin is poured out. This causes agglutination of the two contiguous peritoneal surfaces and is one of the first steps in the repair of peritoneal traumatism. Soon there forms in this layer of fibrin, from a multiplication of subjacent connective tissue cells, a firm fibrous connection between the two peritoneal surfaces. As time progresses and the cells reach maturity these bands become very strong. In some instances when the damage done the serosa is limited in extent or in action for a short time, a process of retrograde change takes place and absorption of the fibrin and of the mitotic cells takes place promptly before firm establishment of the adhesions occurs.

Restoration of the intestine to its normal state soon permits the resorption of this material. Under prolonged exposure, however, this material may remain for longer periods of time. Such agglutinations are, as a rule, only temporary. In cases where the damage is more severe the process of repair goes on in the usual way by the formation of fibrin bundles along which the cells glide.⁶

Permanent adhesions do not follow upon this process unless the irritation is continued, as by the presence of a foreign body, bacterial flora or some substance capable of exciting mitosis of the fixed tissue cells, both the endothelial and connective tissue cells.

The distinction between the processes of agglutination and the formation of permanent adhesions is one of degree and not one of kind. The changes, both cellular and vascular, are identical with those which occur in normal repair. The reason for the persistence of these structures lies in the prolongation of the irritative process until vascularization of the new connective tissue takes place and the surface becomes covered with new endothelium. Under these conditions a tendency to persistence of the new tissue is present.

At abdominal section during the activity of an acute peritonitis the tissues are soft, the intestinal walls are thickened, edematous, friable, and the coils are held together by soft friable adhesions.

When observed at a later stage the intestinal wall has regained its normal tone, the adhesions are extensive, less vascular, but perhaps still soft and are separated without great difficulty. The points of cleavage bleed less freely than during the early stage.

At a still later time when the inflammation has been quiescent for

some weeks, the adhesions are so firm that it seems almost possible to lift the patient by them without rupture. This is particularly true in certain forms of inflammation of the adnexa.

These adhesions are formed of connective tissue with few blood vessels, and a layer of endothelial cells similar in every way to normal peritoneum covers their surface. Such adhesions contract somewhat and are in time diminished in extent by the power of resorption, but may persist permanently. In the absence of continued irritation or useful function, many such adhesions disappear entirely in the course of years.

Sometimes the adhesions are seen as very thin delicate cobweb-like membranes; at other times they are thick, strong, and dense, contracting very markedly. After the reparative process has subsided or any inflammatory action has been checked such adhesions may become attenuated by the pull of intestinal peristalsis. In still other cases there is a slow but constant tendency to clear away the scaffolding which these bands represent, and the part is restored to normal. It has often been the source of surprise how dense and how numerous are the adhesions found at operation performed for inflammatory lesions and how completely they have disappeared at a subsequent abdominal section.

Uyeno¹⁷ claims to have established abdominal adhesions by using iodine solutions in rabbits, and after assuring himself of their presence at a second operation caused their disappearance by massage of the abdominal walls.

Uyeno reviews the histologic work on the subject, quoting various investigators. Heinz believes that adhesion of the serous surface occurs when, for some reason, the endothelium has been lost, and that connective tissue then lies directly in contact with connective tissue. Graser is of the same view, *i.e.*, that from the loss of the endothelial layer proceeds adhesion formation, and that integrity of the endothelium is a safeguard. Ziegler also subscribes to this view.

On the other hand, Muscatello believes the essential to adhesion formation is, first, inflammation of either one or both surfaces, with consequent formation of fibrinous exudation, which is then followed by loss of the endothelium.

This condition becomes more complicated when viewed in the light of opinions concerning the development of fibrinous exudation into the connective substance. According to some¹⁹ the cells underlying the fibrin constitute the endothelium, so that the fibrinous exudate is formed by a "sweating out" of plasmic fluid from the vessels. The contrary opinion, as held by Virchow, Buhl, Grawitz, Busse, etc.,¹⁹ places the

endothelial cells over the fibrin, considering the latter as a fibrinous degeneration. It is thus not clear whether the fibrin in fibrinous inflammation of the serosa is of exudative origin or due to fibrinous degeneration of the connective tissue.

Histologically, Uyeno's findings give the following picture: The fibrin deposit, which represents the adhesive layer, is already infiltrated by the seventh day with large fibroblasts of varied forms. After fourteen days the adhesion tissue consists of fibroblasts and fairly regularly arranged connective tissue fibrils, which, by the eighteenth day, are very definite. After twenty-one days there are variously formed cells and fibrin layers generously dispersed through the adhesive tissue, and by the twenty-fourth day the connective tissue is fully formed, consisting mostly of parallel fibers. He assumes from this that in the inflammatory changes of the serosa cell proliferation comes to a standstill in about ten to fourteen days, and that in eighteen to twenty-four days almost complete involution takes place. So far as the fibrin deposit is concerned, it becomes granular, dissolves and is resorbed.

During this process of new formation of connective tissue and resorption of fibrin, the fibroblasts gradually give rise to tissue fibrils. The fibrin mass which is abundantly present on the seventh day, gradually diminishes, being present in meager bits after fourteen days, and then disappears altogether.

With regard to cellular infiltration, he observed moderate dispersal of lymphocytes only during the first seven days. Polynuclear wandering cells were present in very small numbers for the first twenty-one days. In the first stages of the inflammatory process, leukocytes were more plentiful, but they were scarce in later stages.

The effects of iodine, acids, and alkalies appeared to involve only the most superficial layers. The underlying tissues—muscle and mucous layers—never showed any changes, with the exception of longitudinal muscles which were rarely obliterated by connective tissue.

In his work he found that the increase of elastic fibers goes parallel with that of the fibroblasts. The extensive development of elastic tissue in adhesions is very obviously conditioned by its mechanical extension and expresses a histologic need.

Summarizing, he maintains that the adhesions caused by iodine, acids, and alkalies present about the same picture.

So far as prevention of adhesion formation goes, experiments were carried out on rabbits as in the preceding. Massage was instituted in from twenty to forty days after adhesion formation had taken place. In all experiments, whether early or late, detachment of the adhering

surfaces was obtained, though incomplete in some instances. Uyeno says that it is impossible to judge just how applicable this method is for human beings. He had no results from physostigmin and its effect on peristalsis.

The reparative type of adhesions differs materially from congenital structures. Their vascular arrangement is irregular. The points of contact are different, notably attachments of the omentum, the sigmoid and the rectum. They are often absorbed, while the congenital structures usually remain in situ unchanged.

The early steps in the development of adhesions by the formation of fibrin are not so very different from coagulation of the blood, and the leukocytes may have a function as enzyme carriers which is not unimportant.¹³ The mere presence of blood in the peritoneal cavity does not favor the formation of adhesions according to Schrunder. No adhesions formed even when the serosa was injured at the point at which the hemorrhage occurred, but they did form very readily when microorganisms were added to the blood.

The causes most effective in the production of adhesions are traumatism, infection, the presence of foreign bodies, drainage tubes, glass or rubber, gauze or sea sponges, irritating solutions of various chemicals. Certain forms of bacteria are more prone to produce adhesions than others. *Bacillus tuberculosis*, *pneumococcus*, and *gonococcus* are very active agents in the formation of adhesions.

Causes of the Formation of Adhesions.—Traumatism is generally recognized as a very important cause of the development of peritoneal adhesions. The reasons for their occurrence as the result of open wounds and particularly perforative intestinal wounds are so evident as to excite but little comment. It is not generally recognized, however, that abdominal trauma without a manifest open wound not infrequently results in the formation of troublesome adhesions. Blows from blunt objects, as a large hammer, a cart wheel or similar violence may excite sufficient reaction to produce the formation of adhesions even when there is no evidence of an intestinal leak.

While the determining factor in adhesive peritonitis under these conditions is likely to be damage to the intestinal wall sufficient to permit bacterial invasion, it cannot be denied that cases do occur in which adhesions are formed without any evidence of such a leak.

In view of the evidence it seems logical to conclude that in closed injuries adhesions rarely form in the absence of bacterial contamination from the gut. It is a well-known fact that intestinal rupture can take place with but little clinical evidence of such injury during the early

hours. The presence of late symptoms of localized peritonitis points strongly to a damaged viscus with bacterial contamination. Accidental open wounds often carry sufficient bacterial flora to excite an exaggeration and prolongation of the process of repair so that adhesions may result.

Surgical operations for various lesions are one of the frequent causative factors in the development of adhesions. The presence of adhesions at a secondary operation occurs so frequently that it excites no comment. Adhesions may, therefore, form after an abdominal section performed in a perfectly clean and aseptic manner. Such adhesions are neither so frequent nor so baneful as those occurring after operations for inflammatory lesions or from bacterial invasion of clean wounds.

Any damage to the endothelial layer of the peritoneum may permit the formation of adhesions. Likewise, any raw stump of omentum or any free peritoneal edge may become agglutinated to an intestine or to the parietal peritoneum. The omentum has a remarkable habit of insinuating itself between the edges of an inaccurately closed wound. It has been observed to reach into the small needle punctures made in suturing the peritoneum even when not caught in the grasp of the suture.

Particular emphasis is called to the prompt formation of attachments when the omentum or the gut wall is caught in suturing the abdominal wound.

The readiness of this adhesion formation from simple trauma has been utilized in the Talma-Morrison operation for cirrhosis. Healthy peritoneum placed in contact with broken peritoneal surfaces readily becomes adherent. This is particularly true when the raw pedicle contains inflammatory tissue. This occurrence is not infrequently in excision of the uterine adnexa for suppurative disease.

The importance of bacterial invasion in the formation of permanent and serious adhesions can scarcely be overstated. Simple trauma produces a prompt attachment of the peritoneal surfaces with repair and early resorption. On the other hand bacterial contamination results in the addition of a continuing inflammatory process to the traumatism with an increasing tendency to permanency in the adhesions resulting. Certain bacteria have a much greater tendency to produce adhesive formations within the peritoneum than others. These organisms are usually less active pus producers than the staphylococci, the streptococci, or the colon group. They are, however, none the less persistent and harmful. Among such organisms may be mentioned the gonococcus, the pneumococcus, and the tubercle bacillus. These organisms are notorious for their tendency to form dense adhesions. They excite a larger amount

of cell reaction than the other group which produces a greater circulatory disturbance.

Many observers have called attention to the pronounced fibrin-producing tendencies of the gonococcus, the pneumococcus, and tuberculosis bacillus. This tendency seems to result from their low pathogenicity in a somewhat abnormal habitat. It may also be due to their failure to elaborate a peptonizing principle in sufficient amount to liquefy the tissues.

Attention has been called repeatedly to the tissue reaction resulting from the presence of a foreign substance remaining in contact with the peritoneum.

Nonabsorbable suture material, glass or rubber drainage tubes, gauze or sea sponges, gauze packs, and wicks all tend to cause a persistence of the inflammatory or reparative process and the formation of marked and more or less permanent adhesions.

Any chemical substance capable of injuring the endothelium or the peritoneum may excite the formation of adhesions. Strong solutions of iodine excite a marked adhesive reaction. Hertzler⁶ says that, "corrosive chemicals are less likely to cause adhesions, because the basement membrane is destroyed." It does not seem to the writer that this contention is sound.

Adhesions are not infrequently present in connection with certain tumor formations, particularly ovarian cysts, uterine fibroids, etc., even in the apparent absence of microbic infection.

During the days when tapping of ovarian cysts was done, adhesions were often seen. Traumatic rupture of ovarian cysts or torsion of the pedicle often resulted in adhesions. Similar results occur from degeneration of a uterine fibroid.

Absorption of Adhesions.—Numerous reports appear in the literature which positively show that reparative and inflammatory adhesions have disappeared.

Hertzler⁶ concludes that agglutination of peritoneal surfaces may occur without destruction of the endothelium, but that in the formation of true adhesions the endothelium is always destroyed. If the basement membrane is not destroyed, he believes that the adhesions may later separate. When this membrane is destroyed the union becomes permanent. Such result is shown when coils of intestine become intimately united without the intervention of any tissue.

Proper understanding of the physiological importance of adhesive formations in healing of peritoneal traumatism and the restoration of its structure after inflammatory reactions enables an observer to recognize

their pathological importance. The simple fact that an adhesion is present by no means indicates that it is pathological. Adhesions are pathological only when they cause angulation of the intestine or produce sufficient pressure to interfere with the fecal flow or to cause obstruction to the intestinal circulatory apparatus. Any interference with the normal mobility of an organ by adhesions is pathological.

Adhesions are frequently pathological without producing serious clinical evidence of their presence. They are potentially capable of producing ileus, but the latter condition only results when dietary indiscretion or some other factor causes distention of the intestine so that the band or adhesion by pressure causes complete obstruction.

The changes taking place in adhesions as time passes consist in contraction upon the one hand and attenuation or stretching upon the other. Close adhesions between two adjacent coils may contract and the resulting scar tissue be very firm, showing a considerable amount of mature connective tissue in its structure. The greater the production of fibrous tissue in the adhesions the greater the tendency to contract. On the other hand the smaller the amount of connective tissue stroma in the new issue the less the contraction and the greater the tendency to stretch. Attenuation of the newly formed tissue seems to occur most frequently when the band is attached to the small intestine. The contention seems reasonable that the mobility of the involved loop is an important factor in such attenuation.

Lawson Tait reasoned that by keeping the intestinal contents moving by sulphate of magnesia and by the stimulation of peristalsis after abdominal section that the inflammation of the peritoneum was lessened and the formation of adhesions diminished. This method has been abandoned. It is the consensus of surgical opinion that after abdominal section, and particularly in peritonitis, catharsis is not indicated.

In the absorption of adhesions time is perhaps the factor of greatest importance as shown by the large number of observations made where extensive adhesions found at the first operation have entirely disappeared at a later one. These adhesions do not disappear by magic, but certain factors are active in their resorption.

There is always a tendency of tissue to undergo atrophy when it has no functional importance or is not kept nourished because of irritation.

The most plausible explanation of the phenomena of resorption appears to be contraction of the connective tissue, thus diminishing the vascularity and nutrition of the new tissue, aided by the phagocytic action of certain cells. The exact nature of this process is not fully

understood. It appears that certain large cells, as macrophages, have the property of taking up material from the new structure and perhaps that of producing an enzyme which causes a solution of the connective tissue. At any event when the local irritation is small there is a slow but gradual disappearance of the new tissue. "The processes concerned in their removal consist of degeneration and absorption. What part enzymic processes play in the solution of such adhesions is not known with certainty."¹³

From clinical and experimental observations it may be concluded that adhesions are not necessarily permanent but may disappear.

The removal of adhesions *in vivo* depends upon a process of degeneration with absorption of the detritus. Undoubtedly the mobility of the intestine has an important bearing upon this process.

Deleterious Effects of Permanent Adhesions and Bands.—In the consideration of this portion of the subject the treatment of congenital bands and membranes will be included because of the close similarity of their action, and also of the measures employed for the relief of symptoms produced by them.

The mere presence of an adhesion does not signify that it is productive of any pathology. In fact, it may be entirely innocuous or in some instances serve a useful purpose. It is only when such structures cause interference with normal intestinal function that they become pathological.

One of the most troublesome types of adhesions is that in which the great omentum is attached to the pelvic structures or to the abdominal wall in such a manner that constant traction is made upon the stomach. This produces ptosis and fixation to a marked degree, impairing its motility and its ability to empty itself. The relief following the release and repair of such adhesion is prompt and complete.

Similar pressure and traction may occur from bands affecting the colon or the small intestine which may be sectioned with very happy result.

As an exciting cause of stasis and of ileus the position and attachment of an adhesion is much more important than its size and extent. A very small slender tense band extending from one intestinal loop to another or from the intestine, omentum, or mesentery to the parietes is much more likely to produce obstruction than very broad and extensive adhesions binding a mass of intestinal loops together.

Permanent adhesions result from the completion of the process of repair for the restoration of the tissues following damage due to trauma or infection. They are in reality cicatricial structures. For a con-

siderable time a diminution in size and extent is observed, but finally both remain unchanged. Constriction, angulation, or malposition of the hollow viscera as the result of adhesions are pathological and productive of functional disturbance, discomfort, and actual pain. The importance of adhesions varies with the amount of the disturbance, from the slightest inconvenience and toxemia to complete ileus, and perhaps death of the individual. Some of the milder types of discomfort result from the simplest form of band extending from the parietal wall to the intestine, stomach, or bladder.

In such cases, as long as the viscera are not distended no evidence of the presence of an adhesion is given. The presence of distention pro-

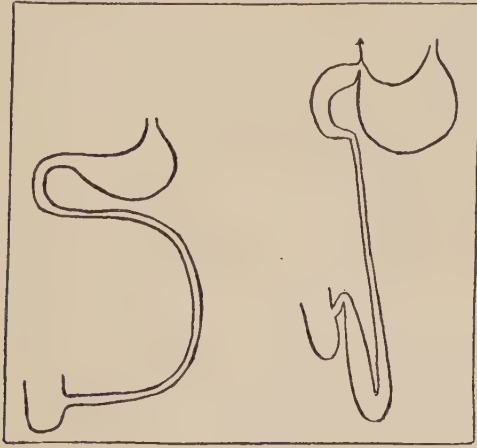


FIG. 41.—A. DIAGRAMMATIC, NORMAL ARRANGEMENT. B. DIAGRAMMATIC, SHOWING TUG OF PTOSIS, CAUSING STASIS. (After Lane.)

duces discomfort by causing traction upon the organ and upon the parietal wall. This is temporary and disappears as soon as the distention is relieved. The evidence of this pull is transmitted to the brain by the nerves supplying the viscera and the parietal peritoneum. A more marked effect is produced by transverse pressure upon an intestine by a band which causes partial occlusion. Such partial obstruction may occur intermittently or may be more or less constant in its action. The grade and continuance of such obstruction depends upon the extent of the adhesions and the narrowing of the intestinal lumen which results.

In the larger number of instances the adhesions occlude the intestinal lumen partially or completely without serious interference with the vascular supply and give rise to intestinal stasis. In other cases the bands by pressure on the distended intestine cause interference with

the circulation to the intestine resulting in necrosis. This condition is met clinically in acute ileus and is very grave.

The effect upon the intestinal wall of the mildest type of obstruction by a band is shown by distention of the proximal coils with changes in color depending upon the amount of circulatory disturbance. Peristalsis is always active in the proximal bowel except in those cases due to acute peritonitis preceding the formation of the band or that resulting from its effect.

The terminal result of complete constriction of the mesenteric circulation by a loop of intestine is necrosis of its area of supply. The portion of the intestine distal to the side of the obstruction is flaccid.

When peritonitis is present in obstruction of this type the lower loops may show some distention, but never to the degree observed above the constriction.

The distended loops in acute internal strangulation lose their power of contraction, become greatly distended and markedly edematous and softened. The contents of such an intestine are very toxic, particularly so when the obstruction is complete.

The seriousness of adhesions varies greatly and depends upon the completeness of the obstruction. In the absence of complete blocking the patient may be able to carry on for years. The condition is mechanical and relief can be afforded only by operative intervention. A fair amount of comfort may be obtained from careful habits of life.

The symptoms of intestinal adhesions depend upon the site, extent, and character of the abnormal structures and upon the amount of interference with the fecal flow. Necessarily because of the great complexity and extent of such adventitious bands and the large number of contributing factors, the symptoms resulting show great variety. Individual reactions also play an important rôle in determining the amount of complaint. In many cases the symptoms only excite a suspicion of intra-abdominal pathology, while in others the attention is quite definitely called to adhesions as the causative factor. The history of a previous attack of peritonitis, an abdominal trauma, an attack of obstruction, of appendicitis or cholecystitis calls the attention to the possibility of the presence of intraperitoneal adhesions.

These patients, if not neurotic in the beginning, become so in time. They suffer from occasional attacks of toxemia, lasting from one to several days, occasionally relieved by a brisk purge. Headache is not infrequent. Lassitude and anorexia are usual. The skin is muddy with a tendency to perspiration in the axilla and in the palms of the hands and soles of the feet.

The onset of the symptoms is gradual in most instances. Some patients can give a very accurate date for the inception. In others it is very vague. Cases of congenital origin usually show some evidence of disturbed alimentary function throughout life. Occasionally added to this history there is a gradual increase in the discomfort produced by the partial stasis. The careful elicitation of such a history will aid in differentiation between the congenital and acquired type of adhesion.

Constipation is present in every case. Occasionally a temporary diarrhea occurs. Mucus may be passed. The amount may be exaggerated if large and frequent doses of drastic cathartics are administered, also in cases where a persistent colitis accompanies the condition. Gaseous distention is always in evidence. Tympany and doughiness are often marked. Belching is of frequent occurrence, and it may be accompanied by regurgitation of food, rarely by vomiting unless the obstruction is considerable.

Persistent nausea, vomiting, and distention are present in obstruction. Loss of weight and of bodily tone from the toxemia is often observed. The mental picture accompanying the toxemia is characteristic. Hebetude, lassitude, depression, marked irritability, and nervousness are usual. Fermentative dyspepsia difficult of control may first bring the patient under observation. Pain is present in every case. It varies from a superficial hyperesthesia relieved by deep pressure to a very acute colicky type. It is rarely constant, occurring rather with attacks of gaseous distention. It is often relieved by emesis. The pain may be referred to any portion of the abdomen, but its most frequent site is the right side. The physical findings show distention of the colon which may be fixed in a malposition, tympany over the small intestine and increased gastric resonance. Frequently inspection shows peristaltic waves in a number of coils while others are inactive. This sign is valuable in determining the site of the adhesion. Borborygmus is frequently audible at some distance and is quite annoying to the patient. On auscultation evidences of peristalsis are elicited. Occasionally masses are palpable within the abdomen. These, in the absence of complications, are often fecal and change in shape from time to time.

Fluoroscopic readings with abdominal palpation give considerable information. A complete radiologic study frequently clears up the diagnosis.

Methods of Prevention of the Formation of Adhesions.—The fact that a large number of measures have been proposed for the prevention of the formation of adhesions following operations demonstrates the difficulty of obtaining such result. The surgical profession

has given a great amount of study to this phase of the subject. The fact that normal repair is accomplished by the production of adhesive material as a preliminary step to the development of new tissue should be given due importance in a study of this subject. The object of the surgeon is to utilize the natural processes of repair and to attempt to keep the changes within normal limits. When this is accomplished, the process remains a physiological one.

It has been apparently overlooked by many workers in this field that overzealous efforts to prevent adhesions to a very large extent have been responsible for their formation. Too little dependence has been placed upon the natural reparative powers of the individual. On the other hand too much dependence has been placed upon the efficacy of foreign substances as preventive measures.

A very large number of substances have been employed in the effort to prevent the formation of adhesions. Among these may be mentioned normal saline solution, sodium citrate solution, albuminous substances, as white of an egg, gum arabic mixtures, gelatin, liquid paraffin, various oils, Cargile membrane, goldbeater's skin, protective silk, fascial layers, and other foreign material.

As early as 1886, Mueller¹⁰ proposed the use of normal saline solution within the abdomen for this purpose.

In 1889 Stern¹⁵ reports upon its use in two animals and adhesions occurred in both, so extensive in one that he considered the solution as directly responsible.

Vogel¹⁸ made several such experiments but failed to prevent adhesions.

Citrate of sodium solution was later employed under the conception that it possessed some solvent action which would prevent the development of adhesions.

The failure of both these solutions to give the desired result may be attributed to two factors, the rapid osmosis which takes place and an interference with the normal physiological reactions. Of all the foreign materials which have been advised these solutions have been the least harmful.

Vogel¹⁸ experimented on rabbits with egg albumin, a mixture of mucin and gum arabic. He concluded that the latter mixture gave the best results. The theory of the action is that the peritoneal surfaces are permitted to glide on each other without friction.

Sterile olive oil was employed by Martin⁷ of Berlin, but his results were not encouraging. The complete absorption of the oil was slow and Wegener²⁰ found several weeks necessary for its complete ab-

sorption. Stern,¹⁵ Bibergeil and Busch¹ found that olive oil is irritating and that it does not prevent adhesions.

Paraffin was employed by Stern¹⁵ but he did not succeed in making it adhere to moist serous surfaces. Busch and Bibergeil¹ found that when liquid it caused an intense irritation of the peritoneum and increased the formation of adhesions. In solid form it acted as a foreign body and was not absorbed. Their observations concerning agar and gelatin were negative. Tallow and fat as applied by Stern and others proved unsatisfactory. Gellhorn⁴ found lanolin to have the same objections as Cargile membrane. Nonabsorbable material such as colloidion, sterile oiled silk, and like material proved a failure.

Morris⁹ proposed the use of a membrane prepared from the peritoneum of the ox, brought to his attention by Cargile of Little Rock. He concluded that in rabbits and in human species this Cargile membrane prevents the recurrence of adhesions, regularly but not completely. The experiments of Craig and Ellis² warrant the belief that neither the chromicized nor the nonchromicized Cargile membrane is of value in preventing adhesions within the peritoneal cavity. The use of thio-sinamin (fibrolysin) is commended by some writers, U. Genino⁶ and C. Michael,⁸ and condemned by others, as Offergeld.¹¹

Cubbins and Abt³ state that from experiments on dogs, "It is very easy to determine by very delicate applications of the half strength tincture of iodine that it destroys the endothelium immediately, and in conjunction with a moderate amount of trauma results in diffuse fibrous adhesions."

Four drams of 3½ per cent tincture placed in the peritoneum with one dram of pus, after being allowed to stand for thirty minutes resulted in death from diffuse general peritonitis in sixteen to twenty-four hours. They conclude that iodine is an intense irritant to the peritoneum and favors rather than inhibits bacterial action.

A careful survey of the literature studied with clinical experience forces the conclusion that no foreign material placed within the abdomen has any beneficial effect in the prevention of adhesions.

The method of viable grafts was first proposed by Nicholas Senn,¹⁴ in 1888, to cover peritoneal defects and prevent adhesions. A modification of the method was afterwards employed by Edward H. Richardson,¹² of Baltimore, which consisted of the utilization of a split portion of the mesentery to cover denuded peritoneal surfaces. The viable graft is far superior to any form of foreign material.

It is now recognized that in the prevention of adhesions and the restoration of peritoneal tissue to normal, dependence must be placed

upon the natural mechanism of defence. More vigorous efforts to aid this mechanism beyond the minute attention to cleanliness and careful technical detail are hurtful.

Dealing with Adhesions.—Undoubtedly one of the most important factors in abdominal surgery in the production of adhesions is rough handling of the intestines and their prolonged exposure to the air. There is nothing in a surgeon's work which measures his skill better than his method of dealing with the peritoneal structures. Rapid, well planned, dexterous work, completing each step of the operation in regular sequence with the least possible insult to the tissues, lessens to a very great degree the postoperative complications.

The surgeon who is indifferent to the amount of damage done to the peritoneum and who leaves a large amount of ragged tissue or who by slow and excessively painstaking work keeps the viscera exposed is exceedingly likely to have a very high percentage of postoperative adhesions and secondary operations for obstruction.

The employment of irritating chemical substances in the peritoneal cavity such as bichlorid of mercury, tincture of iodine, Dakin's solution, alcohol, or ether will greatly increase the number and density of the adhesions.

The presence of infection, particularly when an imperfect peritoneal toilet leaves bacteria with blood clots and dead tissue in the abdomen, tends to result in the formation of adhesions.

These facts should be borne in mind at every operation. Gentleness in manipulation, careful hemostasis, the avoidance of chemicals, and a most careful attention to every detail in technic assure a happy convalescence.

Drains are to be employed only for cogent reasons because a drainage case is potentially a case for adhesions.

At operation for the relief of adhesions or when such conditions are a part of the pathology, when section is done for other affections, congenital anomalies and developmental errors may be corrected with but little fear of reformation. This is true because no inflammatory process exists which will likely cause their recurrence. The peritoneum about such structures is healthy and quickly repairs the damage done by the operative manipulation.

Inflammatory adhesions under similar conditions, however, call for very careful surgery and the separation of all adherent coils with closure of all raw surfaces. Failure to carefully separate adherent coils, or to cleanse localized pus pockets, means failure to prevent the formation of permanent adhesions.

The normal reparative power of the tissue must be fully appreciated to enable the surgeon to carry out the steps mentioned and close the abdominal wound without drainage. This has been done so frequently that its safety is fully proved.

The large number of methods recommended for the prevention of the formation of adhesions clearly demonstrates the difficulty of such accomplishment.

In the author's opinion the best method of preventing adhesions is to keep out infection, to control hemorrhage, and to suture carefully all peritoneal defects after a most careful separation of all existing agglutinations between the peritoneal surfaces. Next in importance is the avoidance, as much as possible, of damage to the healthy peritoneum by sponges or the use of drains. Prolonged exposure to the air and rough handling are to be avoided. Moist gauze sponges are less likely to abrade the peritoneum than dry ones and are, therefore, preferable. Careful suture of the peritoneal layer of the abdominal wall is important to prevent omental and intestinal attachment to the suture lines.

Sufficient emphasis has not been given to the efficacy of rest in the process of repair. We are firmly convinced that by rest the tendency to prompt repair makes for a smaller number of adhesions. Manipulation of the recently inflamed abdomen is not to be permitted.

Every effort should be made to favor the restoration of the general vitality of the patient, since by their inherent power of repair the tissues can best take care of this problem. Most patients are greatly encouraged after operation when they are early placed in a sitting or semisitting position. They are more cheerful, take nourishment better, and altogether their convalescence is most satisfactory.

INTESTINAL STASIS

Interference with the fecal flow is a somewhat frequent condition. It assumes two types; one, chronic intestinal stasis, the other intestinal ileus, or acute intestinal stasis.

Chronic Intestinal Stasis.—This condition is usually of slow development, yet in some instances its cause is present at birth and persists through the life of the individual. In some instances it is not recognized.

Following upon the views expressed by Emil Metchnikoff concerning the toxemias produced by stagnant fecal material in the large bowel and his theory of the prolongation of life by a correction of this con-

dition, much attention was given to the study of fecal stasis and auto-intoxication. Arbuthnot Lane, one of the earliest and most enthusiastic writers upon this subject, brought it before the profession in a most graphic way. He described particularly certain bands extending from the abdominal wall across the cecum and right colon and attached to the lower ileum lying in its mesentery. These bands, he concluded, resisted the strain put on the colon by an overload, and when the colon was prolapsed caused such a kink in the ileum as to interfere seriously with the fecal flow. He became most enthusiastic in his efforts to overcome this stasis, and advised extensive and sometimes dangerous operative procedures for the relief of the stasis and the coincident constipation.

Jabez Jackson, who gave the subject considerable study, described a veil in the right lower quadrant which is known as Jackson's veil or membrane.

One must admit these facts; *viz.*, the function of the large gut is in part that of a reservoir to carry the waste residue of the body and retain it for such time that will permit the second function to be completed. The latter consists of the taking up from the colon the watery portion of the residue and all that remains of nutrient material. Finally it forces the waste from the body at proper intervals.

It is undoubtedly true that the normal power of absorbing nutritive material includes also the faculty of taking up noxious substances which may enter or be formed in the bowel. It appears reasonable to consider that the time required for the absorption of nutritive substances is but short, so that fecal material lying in the large gut for abnormal periods of time is certain to favor the growth of bacterial flora which often produce very toxic material. It is also well established that these toxic substances are taken into the circulation and produce certain well-defined symptoms which disappear very promptly upon taking a brisk purge. In other cases, however, where the bowel carries at all times a considerable residue as the result either of certain anatomical peculiarities, or of defective innervation, the individual becomes a chronic invalid and has a morbid view of life.

The causes of chronic intestinal stasis are any conditions which prevent mechanically the free passage of the intestinal contents. These conditions may be congenital in origin, or, if not developmental in type, are present at birth. Undoubtedly there is a possibility of inflammatory changes occurring within the peritoneum in utero.

It is not always possible to determine clearly whether some of the bands, veils, kinks, and so forth, are really of congenital origin or are due to changes occurring in the peritoneum after birth. Undoubtedly

cases are recorded where the symptoms have persisted from birth in which there is every evidence that the causative factor is of fetal origin.

Congenital pyloric stenosis belongs in this group, although because of its high situation in the alimentary canal there is little evidence of toxic absorption, but the effect is more one of malnutrition, because of the small amount of food passing from the stomach. This condition occurs from the failure of development of the middle coat at the pylorus into mature muscular tissue. The tissue remaining assumes the appearance of a homogeneous, almost gelatinous tissue, but is sufficiently firm to be felt as a resistant structure requiring section to permit the passage of material into the duodenum.

That similar conditions may occur at almost any point of the canal where failure of the structure to develop normally or to rotate properly results in constriction of the intestinal wall. This constriction may not be sufficient to occlude entirely the lumen, but to a degree which delays the fecal stream. The great amount of study in the effort to determine whether a certain band or veil is the result of inflammatory reaction, or is congenital in origin, seems not to have fully clarified the subject, and is really of academic interest. The most important thing to determine for the patient's benefit is whether the bands, kinks or veils are the active causes in the production of the symptoms for which he presents to obtain relief. There are, of course, certain well recognized inflammatory lesions which can be demonstrated as the active factors in the production of stasis.

Tuberculosis of the peritoneum in low grade sometimes runs a very chronic course, produces few symptoms, and causes certain adhesions between the coils, which seriously impede the natural mobility of the intestine, and after recovery takes place leaves fine, delicate but strong adhesions which sometimes completely close the intestinal lumen. In other cases there is just sufficient interference to cause retention with its coincident train of symptoms.

Dowd reports a case of general serositis, which typifies the effects of many plastic adhesions in producing this effect. In this case there was no evidence of tuberculosis. There are other conditions among which may be mentioned Pick's syndrome, producing inflammatory changes within the intestine, which tend to leave adhesions after recovery. This is particularly true in appendicitis and inflammations about the cecum. All forms of peritonitis may leave bands. These are frequently dense at first, but subsequently they become stretched and quite attenuated by the tugging which results from efforts at peristalsis. They may disappear entirely after a time. Often, however, particularly in

pelvic lesions, these thin, apparently delicate adhesions become so strong that the patient can almost be lifted by traction upon them.

Other conditions tending to chronic stasis are chronic tubal inflammations involving and causing thickening of the intestinal wall, inflammatory masses and neoplasms pressing upon the gut.

Partial hernia, or irreducible hernia, or the attachment of a Meckel's diverticulum may produce chronic stasis. When any of these causes are active, the accumulation of fecal material within the colon tends by its

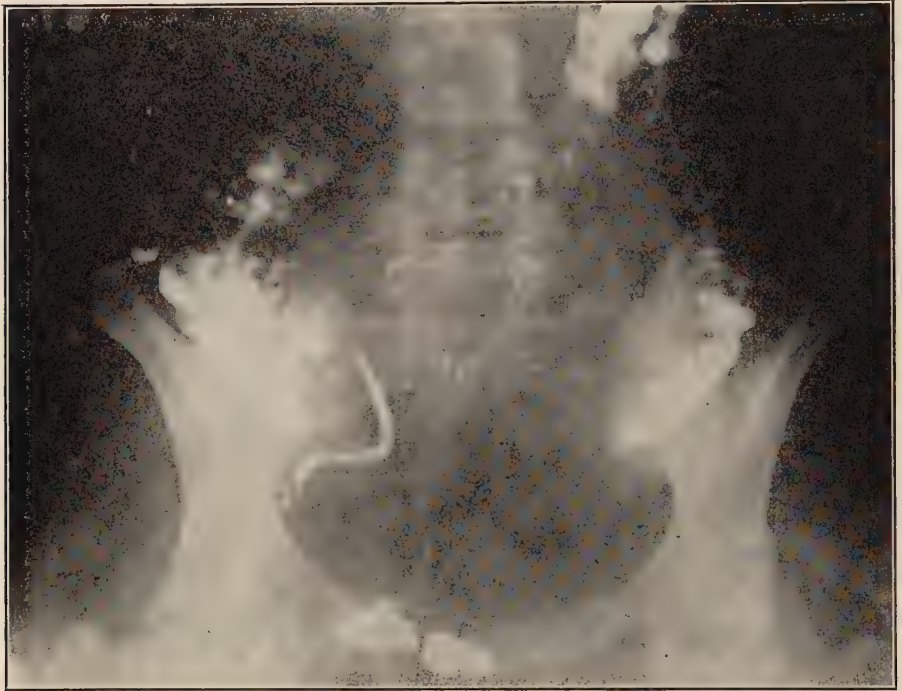


FIG. 42.—MILD TYPE OF STASIS AFTER 30 HOURS.

Notice five fecaliths in lumen of appendix. (Fugate and Enfield.)

bulk to cause sagging of the caput coli, the transverse colon, and also of the sigmoid. By remaining a long time in the intestine it tends to cause abrasions in certain localities, causes distention of the appendices epiploicæ, setting up a diverticulitis and increases thereby the adhesions, and this again delays the fecal flow, making the well-known vicious circle. The patient becomes depressed, lethargic, and does not get sufficient exercise, and this again increases the interference with function. Adhesions of the omentum to the pelvic viscera or cecum following localized inflammations in these localities tends to pull down

the transverse colon so that sometimes it lies in the pelvis and the two loops may become united by bands. This condition is very productive of stasis and is one of the forms amenable to surgical treatment.

There is yet another type of stasis due to imperfect function of the intestinal nervous mechanism, and this is probably most difficult to relieve because the causative factors are not always clear.

The symptoms of chronic intestinal stasis vary very much with different individuals. In some the amount of discomfort is small, and



FIG. 43.—OMENTAL ADHESIONS.

the chief complaint is constipation and the necessity to take increasingly large doses of purgatives. It is really remarkable how some patients accustom themselves to irregularities of habit, and apparently carry on satisfactorily. The truth probably is that they really do not know what it is to feel well. In other active individuals even a temporary fecal stasis produces malaise, headache, and discomfort, demanding immediate relief, which can be obtained readily by freeing the intestine of the toxic material. Where the stasis has persisted for a long time the patient presents as a puny, poorly nourished individual with a swarthy

skin, cold hands and feet, a tendency to perspiration in these parts, marked lassitude and depression.

Arbuthnot Lane describes the toxic symptoms as "headache, a feeling of mental and physical lassitude, an inability to perform the ordinary duties of life, mental misery and distress, nerve symptoms (as migraine, etc.), which are comprised usually under the term biliousness, and a want of control over the temper, which renders these patients unhappy and unpleasant companions."

In one of my own cases the patient, a young woman of twenty-three, had suffered all her life from constipation. She took purgatives daily to get a normal stool. She came under my care for an emergency operation for a ruptured cholecystitis. She had an invagination of the cecum which caused the stasis.

Diagnosis.—This is based upon the history of serious constipation over a considerable period with failure to respond to the simpler methods of treatment. Anorexia, loss of strength and vigor, lassitude, irritability, headache, frequent attacks of migraine, or neuralgia in various regions, tendency to despondency, with cold clammy feet and moist hands, point the way to a diagnosis. A careful physical examination will, by exclusion, bring one to the diagnosis of this condition. It will usually be possible to make out not only the presence of intestinal stasis, but also the most likely causative factor. Careful examination of the abdomen by all the usual physical diagnostic measures must be employed.

As a final aid in clearing up the condition of the intestinal tract the value of radiologic and skiagraphic study cannot be overestimated. In some cases the use of pneumoperitoneum will prove very advantageous.

Prognosis.—The prognosis in intestinal stasis varies very greatly. It depends upon so many factors that the outcome of any given case is difficult of estimation.

The most intractable cases are those in which there is a loss of nervous tone due to innervation with the added dilatation and ptosis which result from prolonged constipation and overloading of the intestine. Such cases require prolonged medical and surgical care, physiotherapeutic measures and a rigorous regimen to obtain any relief at all. Operative intervention in this class of cases is rarely indicated. For the relief of those cases which result from bands, kinks, adhesions just sufficient to delay the progress of the intestinal content, very little can be done by the simpler medical measures. The prognosis for a permanent cure in this class of cases by operation is very good.

The prognosis in all cases of intestinal stasis should be guarded,

since many of these patients are highly neurotic, and even after complete mechanical relief it is often difficult to convince them that they are really well.

In the more severe cases where serious surgical procedures are to be undertaken, there is quite a considerable mortality and this must be given due consideration in making a prognosis in a given case of intestinal stasis.

Treatment.—For the mildest forms, and particularly in the early stages of the neurotic type, where deficient innervation is active, the usual methods for the treatment of constipation are to be employed, with moderate exercise, pleasant surroundings, and careful regimen. Colonic lavage is useful.

When true intestinal stasis of mechanical type is present, the case properly becomes surgical, and in some cases a very remarkable relief follows a very minor surgical procedure.

Lane proposed the transplantation of the lower ilium into the sigmoid and the isolation of the right transverse and left colon, claiming by this step to correct the condition of stasis in many instances. In some he was content to release the bands in the right iliac region, suturing over the denuded peritoneum. He also separated the adhesions holding the two ptotic loops of the transverse colon. In the more severe cases he performed colectomy.

The consensus of opinion at present seems to be that any minor bands and adhesions causing kinks and intestinal stasis are to be handled in the simplest way possible to obtain the desired result. The usual method for obtaining this result is to release all omental adhesions upon opening the abdomen through a sufficiently large incision to obtain free inspection and access. All small bands, veils, or adhesions which are actual or potentially active causes of stasis are severed and all raw surfaces carefully sutured. In many cases minor, non-vascular bands may be separated by the gloved hand and require no suture. In certain cases, when the cecum shows a tendency to prolapse, it is perfectly feasible to shorten the external leaf of the mesocecum with a view of lessening its excursions. In cases where the transverse colon extends into the pelvis and back to its normal site as a double barreled tube with adhesions between the two loops, the adhesions should be severed and sutured over. In cases where it appears that this will not relieve the condition, and in cases of megacolon a resection of this portion of the gut may be indicated. Very rarely is a complete colectomy to be advised. In the very best hands, there will be considerable mortality from this procedure, as shown by Lane's statistics. Therefore, the con-

ditions justifying this operation must be productive of very serious discomfort and distress to justify such a radical procedure. Most of these patients are neurotic, and in many cases complain very greatly after the most careful surgery is done. It is well, therefore, to remember that constipation is a condition which usually responds to careful habit, proper regimen and exercise. It only becomes a disease when distinct pathology can be demonstrated as causative. In the latter condition, sometimes serious surgery is to be considered, and if the cause is removed, the patients obtain great relief. If the surgeon bears in mind that when colectomy or side-tracking prove unsuccessful, this patient has a further handicap to a comfortable existence because of the absence of this important organ, he will weigh the probable outcome well before doing any radical surgery.

Acute Intestinal Stasis or Ileus.—This is one of the most frequent and serious intra-abdominal lesions. It occurs in two forms: mechanical or dynamic ileus, in which there is a mechanical interference with the fecal flow, and adynamic in which a paresis of the intestine prevents the development of sufficient peristaltic waves to carry the intestinal contents forward.

Mechanical ileus or intestinal obstruction is either partial or complete, chronic or acute. In the partial form there is isolation of part of the lumen of the intestine, as in cases of Richter's or Littre's hernia, or where a band or adhesion only partially obstructs the lumen. When the intestine is distended in these cases the obstruction may become temporarily complete, but when relaxation takes place after an enema or gastric lavage, fecal flow may be restored. This condition recurs from time to time until in most cases the obstruction becomes complete. In effect the difference between complete and incomplete obstruction is one of degree and not in kind. There is, however, this exception, and a very important one; *viz.*, there is usually in incomplete obstruction no interruption of the circulation to the entire lumen of the intestine. In a few forms of incomplete obstruction, as Richter's hernia, the small portion of the lumen of the gut pinched by the hernial ring may be deprived of its circulation and ulceration through the intestinal wall take place. The results in such cases are quite like those of complete obstruction.

Complete occlusion of the intestine in almost every instance indicates that there is a mechanical constriction which not only interferes with the fecal flow through a loop of intestine, but also completely occludes the circulation through the blood vessels of the part. From this fact it is evident that every case of mechanical ileus is a serious

menace to the life of the intestine, and if prompt mechanical relief is not obtained the patient's life is jeopardized.

It is impossible to consider fully this subject without a brief mention of the valuable work done recently in the study of intestinal conditions by means of the x-ray. The relation between intestinal stasis and the varying degrees of obstipation is so intimate that it will be necessary briefly to touch upon this phase of the subject before approaching the study of obstruction *per se*. The writer, from a wide experience with necropsy material and at the operating table, as well as by observation under x-ray examination, can positively state, without fear of successful contradiction, that the gastro-intestinal structures by no means have a fixed and certain relation in the abdomen. These relations change with posture, time of day, proximity to meals, amount of food ingested, the amount of material within the intestine, and in accordance with the habit of the patient; moreover, the musculature, development, and attitude of the patient have much to do with the position of these organs. Much has been written, especially since the employment of roentgenology in the study of stasis, concerning various bands and kinks attached to the intestine, and in the opinion of the writer entirely too much stress has been placed upon their importance as causative of interference with fecal flow. Do not understand me to say that bands and kinks do not cause stasis and even obstruction, for they do. I do not believe, however, that any fixed and set bands named by any particular surgeon are more important than other and unnamed structures of a similar type. I do know, moreover, that it is remarkable how much constriction of the intestine by bands, kinks, etc., may occur without interference with the fecal flow. A number of cases of sudden obstructive symptoms have been observed to develop in tuberculous or malignant disease of the intestine where the symptoms had not been sufficient to attract the patient's attention to his condition previously, yet the condition had been of long duration. In some of these cases the matting together of the intestine was so close that it appeared remarkable that obstruction had not occurred sooner. The rôle of fecal stasis as a cause of obstruction brings it within the limits of this study. It is especially the atonic type which tends to result in obstipation, where the large feculent masses accumulate and completely occlude the canal. Undoubtedly with the increased frequency of x-ray examinations and the more prompt treatment of increasing constipation this cause of obstruction will be much less frequent.

The causes of ileus are many and varied. They are best divided for study into those causes acting within the intestine, those acting in

the wall of the bowel, and those arising outside its lumen and acting upon it by pressure and constriction.

1. Conditions within the intestine which produce fecal stasis of more or less complete type are fecal impactions, enteroliths, gall-stones, foreign bodies of various kinds, intestinal parasites, phytobezoars.

2. Those arising in the walls of the intestine are invagination, diverticula, volvulus, ulcers, torsion of the mesentery, strictures, neoplasms of various kinds, malignant disease, tuberculosis, actinomycosis, echinococcus, thrombosis, embolism, paralysis (both from paraplegia and paresis due to local inflammatory infections).

3. Those arising outside the lumen are bands, kinks, adhesions, hernial rings, thrombosis, embolism, pressure from new growths extra-intestinal.

Clinically two forms of this condition present, the acute and the chronic, of which either form may be partial or complete. The acute form may develop without any previous history. The chronic form usually presents a characteristic history but in time is converted into the acute type. Owing to this fact I will reverse the usual order and consider chronic obstruction first.

This variety presents a picture dependent upon the causative factor, which usually gives time for its more careful study and therefore relatively earlier diagnosis. At least the diagnosis can be made in many cases before obstruction is complete and the development of an intense toxemia has occurred, with the exception of those cases in which the first symptom is a sharp attack of complete obstipation. Chronic obstruction most often presents a gradually increasing difficulty to obtain a bowel movement, requiring larger and larger doses of purgatives. As long as the content of the bowel is fluid the movements occur with regularity, but formed masses pass with difficulty and after some days of constipation a temporary diarrhea accompanied with considerable pain and griping occurs. Such symptoms when brought to the attention of the physician should be sufficient to call for a close examination of the abdomen and rectum, the latter being inspected through the proctoscope in the inverted (Hanes') position. If this does not disclose the cause of the symptoms further examination by means of the fluoroscope and the skiagram after a meal of bismuth or barium should be made. By following this plan the attendant is relieved of great possible embarrassment and the patient from a condition which, if neglected, might terminate fatally. Numerous mistakes in diagnosis have in the past been made in the cases of fecal obstruction. Modern methods should reduce the number of such cases to a minimum.

The acute form usually presents as a sudden grave intra-abdominal lesion. It is especially marked by severe colicky persistent pain, accompanied by nausea, vomiting, obstipation, and in some instances with marked shock. The vomiting begins late if the obstruction is low in the canal, and early if in the upper portion, and continues until a fecal odor is perceptible. This symptom corresponds to the findings of Stone, Bernheim, and Whipple⁸ in their experimental work upon obstruction in animals. Meteorism is more marked when the obstruction is low and less when in the upper segment of the gut. It may be so great as to embarrass seriously respiration and cardiac action. Borborygmus is at times marked. The rapidity of development of the symptoms will as a rule be dependent upon the causative factor: temperature is normal or subnormal, respiration is fast and sometimes embarrassed, the pulse is but little more rapid than normal, at first is full and bounding, later it becomes compressible and gradually weaker and more rapid, due to toxemia, pain, vomiting, and loss of rest. Death ensues from exhaustion.

After the bowel below the constriction is emptied, no gas or feces pass if the obstruction is complete. Air injected with enemata may infrequently pass and simulate flatus.

In cases of intussusception severe tenesmus may be present with passages of blood and mucus, and a sausage-like tumor is present. In paralytic types, especially when inflammatory, the most persistent symptom is vomiting, marked tympany and absence of intestinal sounds (so-called still-belly) being characteristic. While in a large per cent of cases diagnosis of intestinal obstruction can be made with fair accuracy, it is often a much more difficult task to determine before intervention the site of the stoppage and the nature of its cause.

Diagnosis.—The diagnosis of acute obstruction depends upon sudden intra-abdominal pain centering at the navel and radiating all over the abdomen, marked tympany, nausea, persistent and characteristic vomiting, complete obstruction of the fecal flow with the exception of the conditions mentioned above, marked borborygmus except in the adynamic form. The absence of fever in the early stages is an important diagnostic sign. Leukocytosis is absent in mechanical ileus until after peritonitis develops. This will prove a very valuable aid in the diagnosis from appendicitis or perforated ulcer.

The following conditions at times resemble intestinal obstruction: Lead colic, acute appendicitis, cholecystitis, general peritonitis, perforation of the gastro-intestinal tract, gall-bladder, or urinary bladder, rupture of intestines from a blow, gall-stone colic, renal stone, in rare instances the crises of locomotor ataxia.

Lead colic comes on gradually; the patient gives a history of working in lead. The abdomen is usually contracted. Obstructive symptoms are not complete as a rule. Vomiting is never severe or protracted. Morphine and atropin may relieve the pain. The blue line on the gums is characteristic. The fact must not be overlooked, however, that the patient may be suffering from lead poisoning and also have a mechanical ileus.

Acute appendicitis can be determined by the acute onset, character of pain, general at first but becoming localized in the right iliac fossa accompanied by tenderness and rigidity. The temperature is elevated, leukocytosis is present, tumor may or may not be found; some gas may pass unless the patient has general peritonitis, and fecal stools may be noted.

Thrombosis of the mesentery very closely resembles the symptoms of an acute intestinal obstruction, a condition which nearly always presents in thrombosis. In addition to the usual signs of intestinal obstruction, the patient presents symptoms of severe shock, which may or may not be accompanied by hemorrhage from the bowel and vomiting of blood. Differential diagnosis is not important because each should receive operative treatment.

General peritonitis usually follows an acute inflammatory disease of the appendix or gall-bladder or some perforation of the viscera, and therefore is often preceded by one or two days' illness. The abdomen becomes greatly distended and rigid throughout. Vomiting is persistent and obstructive symptoms are complete. There is no evidence whatever of peristaltic contraction. The patient's face is pale and pinched and anxious; approaching dissolution is very evident. The pulse is rapid, feeble, and compressible, the skin pale and clammy, breathing is very shallow, of the costal type; temperature is usually elevated, especially if taken in the rectum. Increased leukocytosis is present.

Renal colic is differentiated by sharp pains radiating from the kidney to the bladder and pubes or testis, by retraction of the testis and pain in the back. Pain may cease as suddenly as it began, or may be relieved at times by morphine and atropin. It frequently is accompanied by tympanites and sometimes simulates very closely mechanical ileus. Meteorism is present but there is no fever, and the obstipation is rarely absolute. Blood will be found in the urine. Torsion of a floating kidney presents somewhat similar symptoms and can be recognized by a flaccid abdomen and the location of the tender and mobile organ.

Gall-stone colic follows attacks of indigestion and very often occurs at night. Jaundice may or may not be present. Pain is severe but limited to the right hypochondrium and radiates toward the navel and

around the right rib margin and toward the right shoulder, with tenderness and at times a tumor in the gall-bladder region.

While in the large percentage of cases the diagnosis of intestinal obstruction can be made with fair accuracy, it is often a much more difficult task to determine before intervention the site of the stoppage and the nature of its cause. The location of the obstruction may in some of the chronic cases be determined with certainty by the physical examination of the abdomen aided by the Roentgen ray properly employed. The time of the onset of symptoms, especially of vomiting, the amount of distention and the location of distended coils are especially of value in determining the situation of the obstruction. Vomiting occurs early when the obstruction is in the proximal loops, late when it is situated in the distal segment. High obstruction shows slight tympany, while involvement of the distal portion of the gut causes great distention. The two most frequent sites of obstruction are near the terminus of the ileum (ileocecal coil) and at the sigmoid flexure. The longer the time which elapses from the onset of acute symptoms until the patient comes under observation, the greater the difficulty of accurate diagnosis, owing to the great amount of distention and the muscular rigidity which is likely to be present. In determination of the character of the obstruction, the patient's age enters into consideration. In children one meets intussusception, worms, foreign bodies, Meckel's diverticulum, a constricted appendix, peritonitis, hernia, imperforate anus and, rarely, fecal impaction.

In adults volvulus, hernia, diverticula, bands, peritonitis, foreign bodies and sarcoma are present, while in old patients fecal impactions, enteroliths, gall-stones, hernia, and malignant disease are to be expected.

The entire clinical history is important. A previous operation may be a direct cause, from adhesions. It is well to discover whether he has suffered from hernia or an attack of peritonitis, any intra-abdominal lesion or a blow on the belly.

Examination should be conducted with care, all hernial openings being examined, note being taken of the amount and location of the tympany, also the presence of any dull areas, also of any tumor which may be palpated. The amount of rigidity should also be observed and also the general feel of the abdomen. An empty bladder makes the examination more satisfactory. No examination for suspected obstruction is complete without a digital examination of the rectum, and unless the case is urgent a proctoscope (patient placed in Hanes' inverted position) should be employed. This position has the advantage of certainly locating the malignant or other obstruction in the accessible portion of

the large gut, and in rare instances very soon after the onset it might enable one to relieve an intussusception. With the patient in this position an enema can be given with certainty and celerity. In some cases of the acute type, pain and discomfort may not permit the employment of this method. A very acute onset in an adult, with severe colic, pain, and extreme restlessness, without a tumor, with an increasing tympany and tenderness on pressure, with or without muscular rigidity, accompanied by normal or subnormal temperature and a full pulse of 78 to 80, which gradually increases in frequency, with inability to pass gas or feces, will point to volvulus or a band. If there is a tumor in a well-known hernial region, these symptoms point to strangulated hernia. It is possible, however, for an obstruction in the intestines to occur in a patient suffering from hernia without the latter being strangulated. A more gradual onset, with the history of one or several passages of mucus or blood following an alvine evacuation, with constant pain and a distinct tumor, in a child, accompanied by shock and subnormal or perhaps elevated temperature, points to invagination. In an old patient with a history of constipation of years standing, with gradually increasing pain accompanied by inability to pass gas or feces, and a palpable tumor of doughy type, one may safely assume fecal impaction to be present. However, this form may result from a retention induced by a partial cancerous stricture. If the onset be sudden in a similar patient of constipated habit, with or without a palpable tumor but a history of previous constipation alternating with diarrhea, passing mucus and perhaps blood, and with marked peristaltic wave perceptible over the abdomen, it is a reasonably safe conclusion that the case is a malignant one.

Paralytic or adynamic ileus is usually due to paralysis of the intestinal coils from a peritoneal inflammation, either local or general, in which the motor or splanchnic nerve to the gut is paralyzed. It is accompanied by marked vomiting of dark foul material, by the absence of the sounds of any contraction of the intestine, by great distention or elevated temperature, especially if taken in the rectum, and as a rule with increased leukocytosis, save in the case of a perforating typhoid ulcer.

It is a well-known fact that the most dangerous factor in obstruction, with the exception perhaps of interference with the circulation of the gut itself, is the very toxic material which forms within the lumen of the bowel. This toxic substance forms with the greatest rapidity when occlusion takes place, and it is the cause of death in the majority of cases which terminate fatally. General peritonitis and its toxemia play

a secondary rôle, and these conditions are always accompanied by intra-intestinal sepsis.

Clinical experience has taught the writer that when an obstruction has been relieved it is necessary to empty the canal or the toxemia may kill the patient.

Several plans may successfully accomplish this, but the best is gastric lavage if the intestine is viable and its nerve supply not too greatly damaged. Following upon the relief of the obstruction, prompt evacuation of the bowel will occur and aid in the discharge of toxins. Some operators inject a solution of Epsom salts into the intestine at the time of the operation. This, however, is an unnecessary and a very dangerous practice.

The Lethal Agent in Ileus.—It is very important to consider why intestinal obstruction is so fatal, also what are the most potent factors in the production of a fatal outcome.

Undoubtedly the factor of primary importance in this connection is interference with the circulation to the gut. This seems conclusive, since those cases of partial obstruction and of incarceration in which the circulatory function remains intact do not as a rule terminate fatally, certainly not until interference with the blood supply is added to the interference with the fecal flow does death occur.

The most dangerous forms of ileus are those due to volvulus, a constricting band blocking the vessels, and the complete strangulation of a hernia. All of these conditions are promptly fatal and produce this result, first, because of the destruction of the cells locally by depriving them of their nutrition; second, by the shock produced upon the organism as a whole by the pain, and that resulting from the compression of the nervous mechanism of the intestine; third, because there is always produced within a strangulated intestinal coil a very active poison which is markedly depressant to the circulation. The character of this poison has been studied by Dragstedt and by Stone, Whipple and Bernheim⁸ of Johns Hopkins, and others. The literature is reviewed (R. W. Gerard) in the *Journal of Biological Chemistry* for May, 1922 (55:111). The *Journal of American Medical Association*, Vol. 79, page 1581, Nov. 4, 1922, mentions the work of Dragstedt,¹ and coworkers in this field, and suggests brief review of the subject. All workers agree that the lethal agent is a poison taken up from the obstructed intestine.

Whipple⁸ and his collaborators have shown that the formation of a closed loop of the upper intestine, with both cut ends infolded after thorough washing, and the reestablishment of the remaining intestine

by anastomosis is equivalent to a simple obstruction. An animal with such a procedure dies in a manner typical of obstruction, and in the isolated loop there accumulates a fluid which, when injected intravenously into a second animal excites similar symptoms.

The lethal poisons may be developed notwithstanding the exclusion of the bile, pancreatic, and gastric juices, as well as all foods, and in the absence of any secretion to any particular portion of the intestine. It appears, therefore, that the ultimate source of the toxic material must be the mucosa and its products, or the bacteria. Most experimenters agree upon the above facts.

More striking still are the experiments of Dragstedt,¹ which show that the isolation of a closed loop of the upper jejunum in dogs is always fatal within three or four days, while if the isolated loop is washed and left open within the peritoneal cavity, the dogs survive the peritonitis well. In a few weeks such a loop becomes sterilized, and may be closed. It may become distended and even rupture from a nonpoisonous fluid and cause no toxic symptoms.

"Partial or entire blocking of the blood supply to such a loop is attended by no more serious consequence than resorption of the isolated loop, though the same blocking of the normal intestine is inevitably fatal."³

There has been offered no clear explanation why the early isolation and closure of an intestinal loop is more fatal than the late closure of a similar loop. This could be due to the presence of bacteria, for although the loop was well washed, it could not be made positively germ free, yet it would seem that the experiment was incomplete without cultures taken when the lumen of the loop was washed. Gerard concludes from the experiments of Dragstedt¹ and others that "the contents of an obstructed bowel, then, become poisonous as a result of the formation of histamin and allied proteolytic products by the action of putrefactive bacteria. These poisons produce, on injection, symptoms identical with those of acute ileus. The same toxic fluids introduced into a normal intestine, however, are quite innocuous, and must be, therefore, either rejected by the mucosa or destroyed before reaching the systemic circulation. This has led many workers to postulate of the production of an obstruction poison by the mucosa itself, from which the majority passes directly into the blood, while some is fortuitously discharged into the lumen, whence the toxicity of the fluid."

Whipple, H. B. Stone, and B. M. Bernheim,⁸ of Johns Hopkins, *Journal of Experimental Medicine*, 1913 (17:307), in proof that the poisonous material in the isolated loop was produced by the mucosa, showed

that when the mucosa was destroyed by sodium fluorid, a closed loop did not prove fatal.

Von Zwahlenberg showed that under pressure the mucosa of the intestine undergoes degenerative changes. Undoubtedly, obstruction to its vascular supply and the presence of putrefactive bacteria would increase these degenerative changes. In ileus all the factors are present which are likely to produce these toxic proteolytic ferments.

There seems to be abundant evidence in all these experiments that the fluid from an isolated closed loop may be injected into the normal intestine without toxic symptoms while placed in the blood it is lethal.⁶

This seems to show that the open intestine is able to dispose of certain lethal substances which, when obstruction to the fecal flow is present, react unfavorably upon the organism. The presence of a vascular obstruction seems to diminish the resistance of the animal, and to increase the toxicity of the fluids produced within the intestine. The toxicity of intestinal bacterial flora in the cases of habitual constipation is well known, causing headaches, stupor, lethargy, ill-temper, and depression. This train of symptoms probably results from a small dosage of these or similar proteolytic poisons. A thorough sweeping of the intestines clears the fog away and enables the organisms to function properly.

Gerard states that oxycholesterol, derivatives of glucosamins of mucin, cholin, and neurin from lecithin may be formed and contribute to the toxicity of the loop fluid, but they are not toxic to the same extent or in the same manner as the whole fluid, and therefore are of secondary importance, also that the phenols and mercaptans formed from proteins occupy a similar position.

He concludes that the loop poison belongs in the amin group, that obstruction fluid, histamin, and proteose are almost identical pharmacologically, but that these three poisons show marked differences in chemical reactions.

The latter substances, histamin and proteose, point strongly to a bacterial agency in their production. Bacteria grow readily in the normal and prodigiously in the obstructed intestine, yet clinically the removal by prompt lavage of the fluid delays remarkably the fatal issue, and if the bacterial growth is checked, the fulminant symptoms are not present, and even restoration of the lumen is compatible with recovery.

The absence of toxic symptoms in cases of congenital atresia prior to bacterial invasion has been pointed out by Dragstedt.

The process of dissolution from acute ileus seems to be about as follows:

A sudden occlusion of the lumen of the gut and obstruction of the circulation, more or less complete and synchronous; coincident shock and prostration from pain and reflex nervous mechanism; the rapid production of lethal substances within the isolated loop, with rapid absorption under tension; efforts upon the part of the organism to reject by emesis the noxious material proximal to the obstruction; marked dehydration of the tissues as a result; added burden upon the renal function in the effort to eliminate the poisonous substances which are so rapidly taken into the circulation; progressive prostration, loss of bodily heat, and finally death in exhaustion.

These occurrences in the course of an ileus are so clear in their causation and so deadly in their results when treatment is delayed, and the outcome so happy when it is applied promptly that the surprising thing is that often it appears difficult to bring the patient to prompt operative relief.

Prognosis.—The prognosis is most serious in volvulus, strangulated hernia, obstruction due to bands, embolism, intussusception, and malignant disease. To a large degree it depends upon the accuracy of early diagnosis and the promptness with which surgical treatment is instituted. In the very largest number of cases the cause is mechanical and the fatal termination certain unless relieved promptly, with the exception of certain cases of fecal impaction. Cases of volvulus, strangulated hernia, etc., are among the most grave surgical emergencies if relief is not promptly afforded, but the results of early operation are most brilliant.

Treatment.—The treatment should be prompt and based upon the diagnosis not only of obstruction of the intestines but of the character and cause of the stoppage. The mortality in all forms of mechanical obstruction increases with every hour following its inception, and this obtains even in cases of fecal impaction. This is, therefore, a surgical affection and should be treated immediately by operation when the diagnosis is established. The single type of fecal impaction may proceed to recovery without interference, but there is no certainty of such a result even in this form.

Deaths from intestinal obstruction are due to:

1. Delay on the part of the patient to seek aid.
2. Failure to accept operative intervention until symptoms of impending dissolution are present.
3. Failure upon the part of the attendant to recognize the character of the lesion and its great seriousness and the imperative necessity for prompt interference. If the least possible doubt exists in the mind of

the attendant as to the condition, he should insist upon having a surgeon in consultation at once.

4. The injudicious use of purgatives. If asked when a purgative was indicated in the case of a patient suffering from severe intra-abdominal pain, I should say NEVER; with but rare exceptions to prove the rule this is true. Persistent effort to obtain a bowel movement in these cases by purgation has been largely responsible hitherto for the high mortality rate. The purgative should only be given after the patency of the intestinal tract has been established by the passage of gas and feces per anum. A purgative administered in the case of mechanical obstruction causes increased pain, greater tympany, accentuates nausea and vomiting, and markedly hastens the fatal termination without offering any corresponding benefit. It can be laid down as a maxim that a *purgative can do no good in mechanical ileus, while the failure to employ this measure in simple constipation will do no material harm*. Just one more warning: opiates are to be avoided until the diagnosis is made, since the symptoms may be masked and the attendant misled in his opinion, to the ultimate disadvantage of the patient. It is true that sometimes a dose of morphin, or other opium derivative, with atropin has relaxed spasm and relieved symptoms strongly simulating ileus. Morphin, therefore, may be under certain conditions a diagnostic aid. On the other hand it may, by checking secretions, tend to increase fecal stasis and, too, if it relieves the pain in chronic obstruction, the patient may by its use be induced to defer operation until it is too late to save his life.

Opium, therefore, is to be employed in the suspected cases with due care and deliberative judgment. It is to be remembered that your patient has called you first for relief of pain, and your refusal or inability to give such relief may cause your dismissal and add to the delay which usually obtains before the case comes to the operating table. A case of severe intra-abdominal pain which requires more than one quarter of a grain of morphin for its control, unless in an opium habitu  , is sufficiently serious to justify abdominal section.

The old writers upon this subject dwelt at length upon the use of purgatives, the employment of abdominal massage, tight abdominal bandages, local applications of heat, the hot bath, injections of air into the rectum, change of posture, and the use of various kinds of enemata. Some of these measures are of advantage to meet certain indications and some are worth nothing. Hot or cold packs will at times reduce the size of a hernia and permit of its reduction. The same is true of a hot bath. It would only be proper to employ such measures at the

inception of a strangulation before gangrene could have occurred, especially in an old and enfeebled patient.

A word of caution should be given here concerning the improper and unjustified use of taxis. This measure is always dangerous, especially after four to six hours have elapsed following the occurrence of strangulation in a hernia. There is danger of rupture of the damaged gut and of the reduction of the hernial mass without relief of the constriction. Prompt operative intervention is much safer for the patient. The use of enemata is justifiable to aid in making a diagnosis, but it is very unwise to waste valuable time giving repeated rectal injections in the presence of pain, fecal vomiting, great distention, and complete failure to pass gas or feces. If one or two well-administered stimulating enemata of Epsom salts one ounce, glycerin 2 ounces, water or soap-suds 3 ounces, or one containing one or 2 teaspoonfuls of alum to the quart, or one of equal parts of milk and molasses fail to produce results, a diagnosis of mechanical ileus is justified. Then preparation for operative intervention should be made. In the meantime try what can be done to relieve the pain, nausea, vomiting, and perhaps hiccough. In my opinion no measure to meet these conditions equals the employment of gastric lavage. It gets rid of a quantity of noxious material which is endangering the patient's life and adding to his discomfort. It relieves the tympanites and renders administration of the anesthetic and the necessary surgical steps easy of accomplishment, makes the post-operative convalescence easier and the recovery more likely. Therefore, I urge employment of gastric lavage, and refraining from the use of purgatives in intestinal obstruction. In this way and by promptness in applying surgical treatment, the mortality may be reduced to nil, save in the malignant cases.

Just a few words upon the surgical management of these cases: The following plans are recommended:

1. Early intervention.
2. Free median incision to permit access to any part of the abdomen, unless there is some special reason for opening elsewhere.
3. Relieve the constriction, and remember there may be more than one.
4. Expose the intestines as little as possible, and protect with warm moist gauze.
5. Work quickly but carefully.
6. In the absence of gangrenous intestine, the abdomen should be closed without drainage.
7. Gastric lavage will be of advantage in every case.

8. When distention is marked it will be found advantageous to open the gut and drain away through a glass and rubber tubing the noxious material within the lumen, closing the opening in the intestine by a Lembert or purse-string suture.

9. In dealing with gangrenous bowel or with malignant occlusion, one must decide whether an immediate resection or a temporary enterostomy offers the patient the best chance for a recovery. Of course, the ideal operation is resection, but before it is selected the patient's general condition, age, vitality, and power of resistance must be considered. It is undoubtedly true that enterostomy may be a life-saving measure under these circumstances.

In handling cases of this type every effort should be made to conserve the flagging vitality of the patient. Operative work should be done with dexterity and the greatest speed commensurate with due care. Prolonged exposure of the abdominal viscera and slow surgery often result in a fatality when rapid work with little exposure would save the patient. Decision concerning methods of procedure should be made without delay.

A certain number of these cases may be saved by high enterostomy. This can frequently be made under local anesthesia in a very few minutes without serious shock to the patient. Under these circumstances the surgeon is justified in grasping the first distended coil which presents and suturing it to the abdominal wall. It is usually wise to empty the intestine at once through a rubber tube inserted into the bowel and left in situ. When the patient is successfully carried through the critical stage other operative procedures may be employed.

The writer has concluded from his own experience in obstruction from malignant disease that a preliminary enterostomy will prove of great advantage in many cases and permit the subsequent removal of the growth, when an attempt to remove the latter at the time might result fatally.

Drainage of the abdomen is to be employed only in cases where general peritonitis is marked or gangrenous intestine is present.

INTESTINAL PARALYSIS (ADYNAMIC ILEUS)

Undoubtedly the most frequent cause of this condition is acute peritonitis of the spreading type, due to perforative lesions. This condition has been described under the head of peritonitis and attention called to loss of contractile power in the intestine, inability to pass feces or gas per rectum, marked tympanites, persistent vomiting which is finally stercoraceous. Collapse and death end the scene.

To a lesser degree loss of muscular power in local segment occurs in every case of peritonitis, even of local type. Frequently this is slight and produces but little disturbance. Local traumatism, blows upon the abdomen, prolonged distention from a chronic obstruction or from temporary internal strangulation or a hernia cause temporary loss of contractile power. Ill advised and prolonged manipulation in efforts at taxis tend to produce intestinal paresis. The same is true of prolonged manipulations of the intestine at operation, and this explains why some surgeons find their operative cases complain so much of gas pains and tympanites. Traction on the mesentery or its prolonged compression tend to produce paralysis of the intestine.

Embolism of the superior mesenteric artery soon disturbs the nervous mechanism to the production of marked meteorism. This condition is almost always fatal. Prolonged use of clamps in making a resection is likely to be followed by loss of contractile power in the portions of the bowel between the site of the clamps and the line of suture. This is occasionally an important factor in producing a fatal result. Such clamps should not produce too much pressure and should be released as speedily as possible.

The pathology of adynamic ileus is identical with that of spreading peritonitis. The coils become agglutinated together; they are remarkably ballooned. If released from the abdomen they can be replaced with difficulty. If an effort is made by opening the tube to permit the escape of the fecal and gaseous contents only a limited portion is emptied. This results from the inability of the intestine to contract, or loss of the normal peristaltic power. It is often very difficult following an abdominal section to distinguish between this type of ileus and that resulting from mechanical causes, since in a considerable percentage of cases both factors are active.

The symptoms presented by paralytic ileus are persistent nausea and almost continuous vomiting. The expulsion of material from the stomach is in many instances more of a regurgitation than vomiting. The character of the vomitus is very offensive, yellowish or with slight greenish tinge, quite irritating to the skin. Usually it continues until dissolution unless promptly relieved. The fluid is extremely toxic and depresses the patient very rapidly. There are also marked anhydremia and loss of water from the tissues which cause depletion. The condition of prostration is rapidly increased. The patient presents an alarming picture.

Marked abdominal distention, great rigidity, tenderness, and difficult rapid respiration from the distention are usual.

The symptoms of threatened collapse, cold, clammy skin, sunken eyes, pinched nose, rapid and feeble pulse indicate the fatal outcome. The mentality in some instances remains clear throughout, but in many cases it is obtunded and the patient becomes irrational.

Recognition of Adynamic Ileus.—Recognition of adynamic ileus may be made by a history of traumatism, or of some one of the many conditions which provoke the development of spreading peritonitis. Vomiting of persistent type is present, finally feculent in character. There is failure of feces and gas to pass. Marked distention, cold sweat, great prostration, rapid, feeble pulse with diminished arterial tension are always present in advanced cases. The temperature may be subnormal but usually it is elevated. Occasionally the rectal temperature is high when subnormal in the axilla. These symptoms point strongly to acute, general peritonitis with paralytic ileus. The diagnosis is made certain by the failure to elicit any evidence of peristalsis either by inspection, by palpation or auscultation of the abdomen.

Treatment.—The treatment of this condition is that customary in acute general peritonitis. The patient should take no food. The toxic intestinal fluid should be removed by repeated gastric lavage. This measure lessens the distention, diminishes the toxemia, makes the patient more comfortable.

After the nausea and vomiting improve or cease it is justifiable to use an enema when the passage of gas gives evidence of patency of the intestinal canal. Small frequently repeated doses of pituitrin are recommended to overcome the adynamic condition.

This is one of the most serious of surgical conditions. Every effort is to be made to prevent its development. When it occurs after an abdominal operation the question of reopening becomes one for prompt decision. It is very difficult to determine in some of these cases which will give the more favorable result, expectancy or a second operation. All secondary operations are serious. Therefore the surgeon's opinion must be formed promptly, since tardy measures for relief will be unavailing. When there is strong presumptive evidence that a mechanical ileus exists in addition to the paralytic or inflammatory stasis an immediate section is indicated.

If the patient's condition will not permit the use of a general anesthetic or complete operative measures, enterostomy should be quickly performed under local anesthesia as the best that can be offered. In postoperative paralytic ileus enterostomy is especially good in properly selected cases.

This is not the place for slow surgery. The enterostomy should be

employed as a life-saving measure and completed with the utmost dispatch, since every moment of time adds to the depression.

The opening should be made above the point of constriction. The primary object of the enterostomy is to drain the canal of its noxious material.

In those cases in which the paralysis is due to inflammatory or other injury to the gut and the stasis is not mechanical, the following measures are indicated: Absolute rest aided by opiates used as sparingly as may be to obtain the desired result. Repeated gastric lavage becomes the most powerful agent at hand. Food must be withheld absolutely. In certain instances it will be found necessary to use stimulants. Of these epinephrin solution is probably best. Some authorities decry the use of strychnin, but it seems to hold up a flagging pulse. Other stimulants such as digitalin and camphorated oil may be employed.

The author's experience is that when the patient reaches the stage when vigorous stimulation is necessary to keep up failing vitality in cases of peritonitis with adynamic ileus, the battle is practically lost. If the patient does not respond to the measures outlined above, he will not do so at all.

VOLVULUS

Volvulus is a form of intestinal obstruction which consists of the rotation of the bowel upon its own axis or of a torsion of its mesentery sufficient to produce occlusion of its blood supply.

Fortunately, because of its serious nature, it is one of the least frequent forms of intestinal obstruction. It occurs more often in adults. Fitz gives the highest number in the third decade. Gibson found in 1000 cases of acute obstruction 12 per cent were due to volvulus.

Gibson found that in 121 cases of volvulus in his series of 1000 cases of obstruction, 58 occurred in the sigmoid, 15 in the other portions of the colon and in the small intestine.

Strangulation of the great omentum has been reported in a few instances. Some authors place its frequency of occurrence at 2.5 per cent.

Causation.—The conditions which contribute to the development of this type of obstruction are certain anatomical peculiarities, among which may be mentioned a long, narrow mesentery with a short base, the presence of a band or a Meckel's diverticulum. Other diverticula, acquired bands or other abnormalities may predispose to its development. Constipation with considerable residue undoubtedly favors its occurrence. A loaded bowel in the presence of one of the above

mentioned anatomical conditions may be sufficient to excite its formation. The presence of adhesions may permit one loop of intestine to rotate about another which is fixed. Traumatism, straining and coughing have been reported as causative. When a loop of intestine has been injured and its power of contraction impaired, as sometimes occurs when an incarcerated hernia or one that is strangulated has been relieved either spontaneously or by manipulation, it becomes increasingly liable to this accident.

Pathology.—The mechanism of the development of a volvulus is simple. A heavy loaded sigmoid, for instance, aided by its efforts to empty itself, may by irregular contraction in different portions thrust one loop over another. This segment is carried one half turn around and the distended segment may by pressure occlude the loop beneath.

This rotation may take place in the axis of the intestine itself. The twist of a loop may occur with a portion or the whole of its mesentery as an axis. A frequent method of development is the torsion of a loop about a band or diverticulum as an axis.

The first of these types is sometimes seen in torsion of the great omentum. Similar torsion of pedunculated uterine tumors and ovarian cysts occurs with some frequency.

Some writers claim that a torsion amounting to two-fifths of a circle is sufficient to produce obstruction and disturb the circulation of the part. Lawson claims that 270 to 360 degrees is necessary to produce occlusion. Sometimes the rotation may be carried much farther than this. The more pronounced the rotation and the closer the constriction of the vessels, the more marked are the tissue changes, and the more acute are the symptoms presenting.

Only a few hours are necessary to cause necrosis of the twisted loop. Immediately upon death of this segment, acute peritonitis ensues with its usual phenomena.

Prognosis.—This is probably the most fatal form of mechanical ileus when permitted to continue unrelieved. On the other hand, when recognized soon after its inception and promptly treated, the results are brilliant, and the relief is very prompt.

Diagnosis.—The diagnosis is made from the sudden onset of exceedingly acute intra-abdominal pain, often excruciating in character. The sufferer rolls about in agony, demanding instant relief. He may present symptoms of shock, but as a rule the pulse shows only moderate increase in frequency at first, but it soon becomes rapid and feeble. The temperature is either normal or subnormal. Vomiting is a prominent symptom, occurs early and persists until the bowel is relieved of the

twist. There is marked borborygmus. Considerable distention of the abdomen occurs. The active peristalsis may be made out by the observance of the prominent coils upon inspection. Palpation also may afford similar information, and on auscultation one can readily detect the exaggerated peristalsis.

Once recognized, the treatment is similar to that of acute strangulation. Immediate abdominal section should be made and the torsion of the intestine relieved. In the very early stages this is all that is necessary. If the twist has occurred more than six hours previous to operation the intestine will be likely to become greatly damaged or gangrenous.

After the relief of the volvulus, the color should return promptly in the obstructed loop. The application of hot packs of sterile gauze applied for some fifteen or more minutes will show prompt response in the circulation, if restoration of function is possible. The signs of complete destruction of the vascular supply are maceration of the coat of the gut, softening of the peritoneum and a tendency for it to separate from the muscularis on manipulation. The arteries and veins are filled with clots and the color of the mesentery and the intestine itself remains purplish and black. If the patient's condition is good, it is quite justifiable to allow plenty of time for evidence of restoration of the circulation. The evidences of returning viability in the damaged segment are mottling of the intestine, soon changing into a bright pink. The change is so marked that but little difficulty is experienced in determining that the circulation will be restored. When the color only partially returns, and even though heat is kept applied it remains dark and mottled so that there is doubt as to the viability of the intestine, it is unsafe to replace it.

There are several ways in which such a loop may be handled. If the patient's condition is critical and it is likely that a radical procedure will be fatal, it is well to bring the damaged loop into the wound in such manner as to permit its being opened at once or within a few hours, if the symptoms demand it. Under these circumstances the damaged mesentery may be sutured to the peritoneal wound so the entire loop and its mesentery remains extraperitoneal. That portion of the loop protruding should be covered with sterile gauze moistened with warm saline solution. This dressing should be kept warm and moist by the application of normal saline solution at 110 degrees. Over all should be placed an oil paper or silk covering, and a hot water bag used if necessary.

In placing the loop in the wound it should be arranged so that there is no obstruction to the passage of feces through it. There must also

be provision for the mesenteric circulation. The next plan offered consists in making an immediate enterostomy, after emptying the afferent coil through a Mixer tube. This permits the ready removal of a large amount of toxic material from the proximal loop, and will contribute materially to the recovery of the patient. Enterostomy is undoubtedly a life-saving measure in many cases. Its disadvantage lies in the fact that a secondary operation with its added risk is not a remote possibility. In those cases where the condition of the patient will justify it, undoubtedly the operation of choice is an immediate resection of the damaged portion of the bowel. For this to be successful certain conditions are required. The patient's circulatory and respiratory apparatus must be functioning properly. There must be no extreme shock present. The extent of damaged bowel must be limited and the surgical procedures must be completed carefully, skillfully, and expeditiously.

The proximal intestine should be emptied of its content before the resection is completed as described above. Usually when the entire segment is removed, even though gangrenous, abdominal drainage is unnecessary except in those cases where general peritonitis is already present.

Before leaving the table the patient should be given a thorough lavage of the stomach, unless this was completed before the anesthetic was administered. One of the most serious dangers from operative procedure in all forms of ileus is that of the patient drowning in his own vomitus. Therefore, gastric lavage is imperative as a preliminary to any operative measures in all forms of mechanical or paralytic ileus.

The chief danger following fixation of the loop in the wound and also from enterostomy lies in the possibility of embolism from clots remaining in the mesenteric veins. The possibility of such occurrence must be borne in mind when determining which plan of treatment shall be followed. The fact that the performance of an enterostomy high in the small intestine by either of the methods above described materially interferes with the nutrition of the individual, makes the radical operation of immediate resection preferable in such cases.

Following the operation the usual measures for overcoming shock are to be employed. The most important of these are heat to the surface, morphin for pain, atropin to prevent heat loss and the instillation of normal salt solution into the veins, under the skin or into the rectum. Minute but continuous dosage of adrenalin solution will often prove beneficial in these cases. Very little attention need be paid to evacuation of the bowel, for when the lumen of the intestine is properly restored,

this function returns in due course. Gastric lavage is indicated when there is any distention present.

The loop placed in the first method is to be opened promptly as soon as the wound is able to protect itself from contamination, usually in twenty-four to forty-eight hours. In this method and also in the case of enterostomy, a rubber tube fixed in the afferent loop will permit its evacuation without soiling the wound.

Strangulation of the intestine results from internal hernia, angulation over a band, Meckel's or other diverticulum. It is also a very frequent complication of external hernia. Ptosis of certain portions of the intestinal tract may produce strangulation. The pathological changes resemble very much those of volvulus. While usually less extensive the same vascular changes and necrosis of the intestine result. The symptomatology is quite similar.

The onset is usually sudden, but sometimes there has been some complaint of abdominal discomfort for some time preceding this accident. The symptoms are identical with those of strangulated hernia with the exception of the absence of a swelling in a well-known hernial region.

The diagnosis, prognosis, and treatment of strangulation of the intestine are practically those of volvulus. The only difference consists in the necessity for severing the constricting band and making such repair that there will likely be no recurrence.

INTUSSUSCEPTION

Acute intussusception is one of the most frequent abdominal emergencies in infants. It occurs with less frequency among adults, perhaps one case in adults to ten in children. Bolling gives the frequency in males as twice that in females.

The larger number of cases occur at the ileocecal valve or shortly involve that coil. Second in frequency of location is the sigmoid and rarely it occurs in the ileum.

The writer has observed a very unusual case affecting the ileum, in which after the relief of one invagination and during the subsequent manipulations, a second one occurred.

This case gave an opportunity of observing the intussusception in the process of formation, and apparently the contracting portion of the intestine forces a contracted portion into a flaccid or relaxed segment.

Causation.—Undoubtedly the intestine is more irritable, and its

muscular action less well coördinated in infants, because of nervous instability, than later in life. Certainly perverted peristalsis is one of the ways in which invagination develops. At times it is difficult to account for this altered mechanism. It may result from an irritating toxin within the intestine which upsets its rhythmic contractions, or from irritating bacterial flora in the bowel. A cerebral or spinal toxemia may unfavorably influence the nervous mechanism controlling intestinal peristalsis. It seems probable that the amount of mobility of the cecum and right colon, because of the persistence of its mesentery in infants, may be a contributing factor. About 48 per cent of fetuses at term have a mobile cecum. This mobility continues up to the second year, and sometimes through life.

Injudicious feeding would appear to be a contributing cause, but 41 of Bolling's 50 cases were breast fed. This gives rise to the thought that the deficiency in the calcium content occurring in breast fed babies may have an important relation to the cause of intussusception, by causing tetany.

It is easy to understand that the swelling of the mucosa resulting from irritating substances within the intestine, perhaps making the wall thicker at one point than another, might be a factor. The theory advanced by Perrin and Lindsay⁶ is that the determining factor is the production of the equivalent of a foreign body. They suggest that the foreign body is provided by the swelling of the lymphoid tissue.

The presence of neoplasms is important in the causation of this condition in adults. In Eliot and Corscaden's report, 100 of 300 cases in adults had some form of neoplasm as causative factor. Certainly this cause is infrequent in infants. From the observation in one of my cases where the invagination reformed under our vision, it is not surprising that such phenomena occur, but that they do not occur more frequently. Apparently all that is needed for its occurrence is an excessive spastic contraction of separate segments, while the contiguous portions remain flaccid. Worms appeared as the cause in 3 of 43 cases. Traumatism and straining have been mentioned as causative.

The most complete record of the various causes acting in the production of intussusception in adults is given by Eliot and Corscaden. In 300 cases, 60 were due to traction from benign tumors, 40 were caused by the presence of malignant neoplasms, 30 cases were the result of Meckel's diverticulum. "In 23 of the 300 cases cited some form of traumatism was given as the exciting cause."

Eliot and Corscaden's first table of this series is as follows:

Trauma, not classified	2
Blow	2
Crush	1
Fall	3
Violent muscular movement	
(football, 1; riding, 2)	7
Lifting heavy weight	7
Puerperium	1
Typhoid	5
Dysentery	6
Tuberculosis	5
Simple inflammatory ulcers	3

Stephen H. Watts,⁷ reports two cases of this condition due to neoplasms, one due to multiple polypoid adenomata and the other to sarcoma.



FIG. 44.—RECURRENT ADENOMATA OF SMALL INTESTINE.
(Case of Dr. Stephen H. Watts.)

In one of his cases four operations were performed before a cure was obtained.

At the first operation in Watts' first case the invagination, 25 cm. long, was replaced, August 7, 1906. There were few adhesions and no gangrene. Two tumors of the small intestine and one of the sigmoid colon were recognized.

Seven days after the first operation reinvagination occurred and the ileum for 17 inches was resected.

October 8, 1906, a persisting fecal fistula necessitated the third operation at which four inches of the sigmoid colon including the tumor was excised. The bowel was united by lateral anastomosis.

The fourth operation, January 3, 1907, consisted of the excision of

multiple adenomata of the jejunum through incisions in the wall of the intestine on the side opposite the mesentery.

H. M. McKeithen,⁵ house surgeon, Louisville City Hospital, observed a case of papillary adenocarcinoma occurring in a man of twenty-

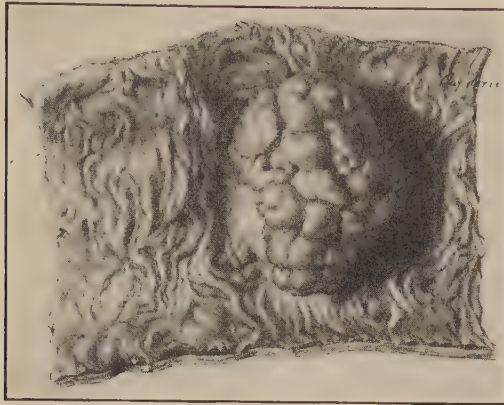


FIG. 45.—ADENOMA OF LARGE INTESTINE CAUSING INTUSSUSCEPTION.
(Case of Dr. Stephen H. Watts.)

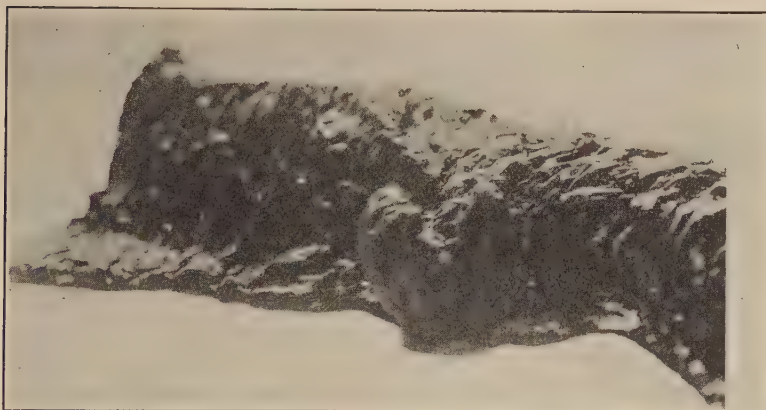


FIG. 46.—ILLUSTRATING PAPILLARY ADENOCARCINOMA OF THE JEJUNUM CAUSING
INVAGINATION.
(Case of Dr. McKeithen.)

six, involving the upper portion of the jejunum, causing an invagination of this portion of the intestine.

The pathology found in the first stages after its inception consists of simple invagination with no interference with the circulation and no swelling for the first few hours, when the amount of invagination involves only 2 or 3 inches of the small bowel,

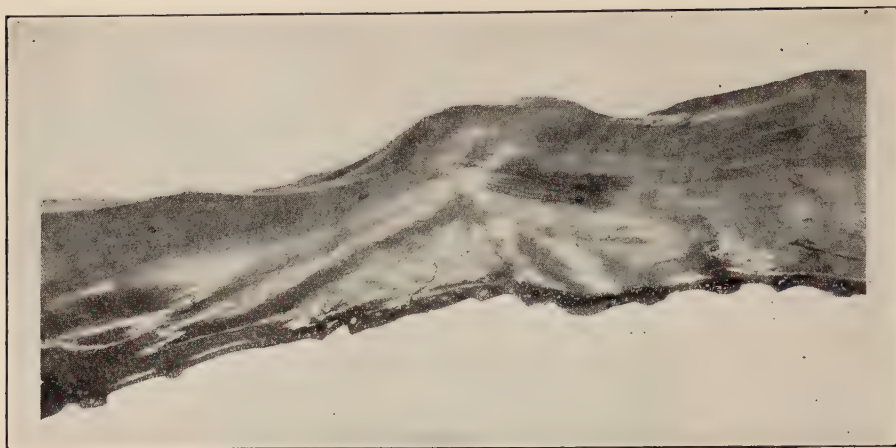


FIG. 47.—SAME CASE SHOWING THE CICATRIX-LIKE CONTRACTION UPON THE SEROSA AT THE SITE OF ORIGIN OF A PAPILLARY ADENOCARCINOMA.

(Case of Dr. McKeithen.)

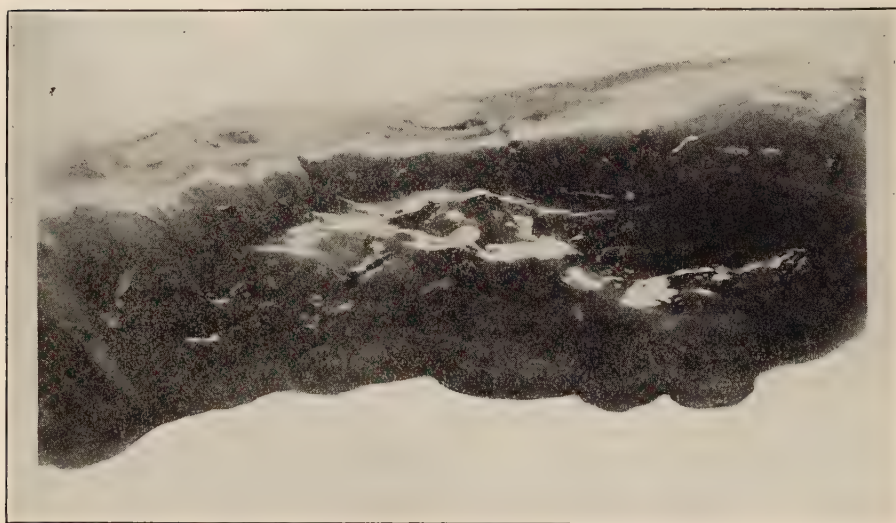


FIG. 48.—MESENTERIC THROMBOSIS RESULTING FROM INTUSSUSCEPTION DUE TO NEOPLASM.

Note the clots in the mouths of the vessels. (Case of Dr. McKeithen.)

In such a case spontaneous reduction may occur. This, however, does not take place with any frequency after the intussusception has progressed to the point of occlusion of the vessels of the intussusceptum. Soon after this occurs, coagulable lymph forms rapidly over the intussusceptum, and fastens the loops together. The swelling increases as the obstruction progresses, and the constriction of the receptum be-

comes so great that necrosis occurs. For some time before this takes place, however, the included bowel is darkly congested, livid, and markedly edematous. There is poured out into the intestine a considerable amount of serum and blood from this strangulated and edematous loop. The mass of bowel lying within the receiving loop tends to increase the spasm of the intestine and thereby to increase the amount of intestine invaginated. Sometimes the intestine for a number of feet is involved.

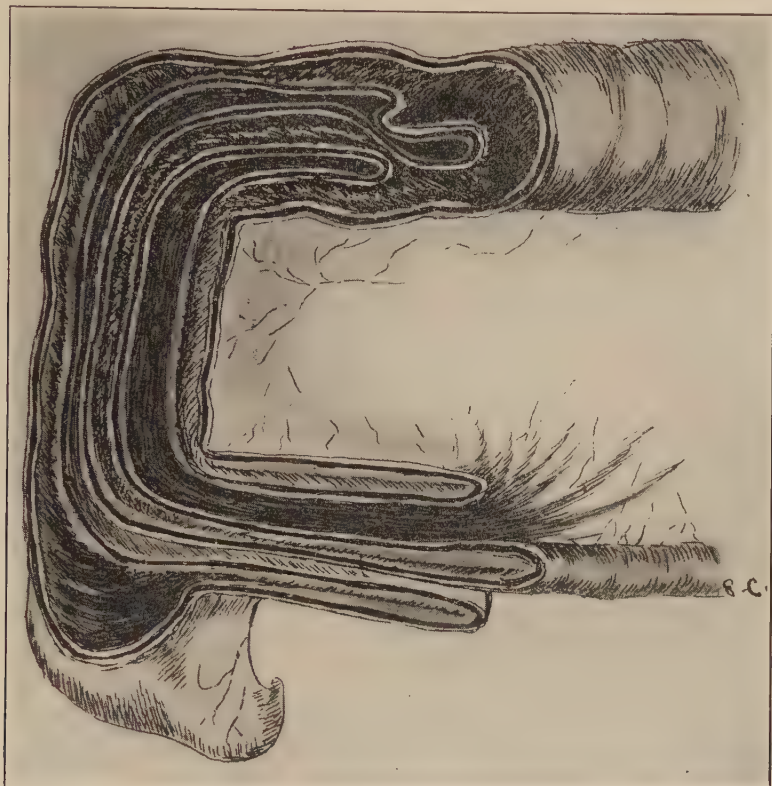


FIG. 49.—INVAGINATION OF THE INTESTINE.
(Redrawn from Coffey.)

The swelling, the constriction at the point of invagination, and the rapid formation of adhesions between the entering and the returning layer render the reduction of the mass difficult and often impossible after the lapse of many hours.

There are on record, however, a number of cases where spontaneous cures have occurred. At least there are a considerable number in which sloughing of the invaginated gut occurred and the patient lived in fair health (43 in 300 of Eliot and Corscaden's cases).

A case of interest came under the observation of the writer in which the patient at two and at seven had attacks which were clearly due to invagination.

When sloughing of the intussusception occurs before the adhesions between the entering and the receiving layers have become firm peritonitis may occur. This may take place at the point of entrance and constriction from a leak subsequent to ulceration. It may also occur at the same site from misguided efforts upon the part of the attendant to correct the invagination by fluids or gas pressure in the colon. It may also occur from bacterial infection spreading through the damaged intestinal wall.

The peritonitis excited by either of these conditions just described is always a serious complication, and adds further mortality to an already dangerous affection. Such a termination can be avoided only by the most prompt and thorough surgery in skillful hands.

The outcome of a given case of intussusception will depend on what measures are primarily employed. It is an essentially dangerous affection. The dangers are: Primary shock incident to the accident, also that due to loss of blood, and that due to pain and tenesmus; absorption of noxious products which are always formed within the intestine in cases where the lumen and the circulation are blocked; acute peritonitis which may follow within six or eight hours from the occurrence of the intussusception; sloughing of the invaginated loop—this dead and decomposing bowel may lie in the colon and from the foul bacterial flora and ptomains it may so depress the patient as to produce death in exhaustion, anemia from loss of blood and toxemia, which may be fatal some time later.

With all these possibilities present it is really remarkable that any case ever gets well after a few hours have elapsed.

Symptoms.—The onset in young children is usually sudden. Frequently a previously healthy child is suddenly seized with abdominal pain and tenesmus, perhaps following upon a large stool. The only discharges are composed of mucus and blood. The latter is sometimes passed in considerable quantity. There is usually no passage of gas. The symptoms are quite like enterocolitis and dysentery.

Careful investigation, however, will in a considerable percentage of cases reveal a sausage-like tumor. This mass is usually mobile and moves with respiration. Under anesthesia, masses which cannot previously be made out are readily felt. The abdomen is usually rather tense with a doughy feel. It rarely presents the extreme rigidity noted in inflammatory conditions.

Shock is often marked and when the hemorrhage is considerable, there is marked anemia.

Even in the acute cases the symptoms are not always typical, but in the majority the classical symptoms are present. In the early hours of an invagination the lower bowel may be emptied, and gas may pass, but after the obstruction is complete, no gas or feces pass. The efforts at stool cause only the discharge of blood and mucus. These symptoms with a demonstrable mass justify a diagnosis of intussusception. This mass may vary in size, depending on an increase of the invagination.

In adults the symptoms vary more than in children, and in many instances there have been symptoms previously of intestinal derangement existing for some time.

Diagnosis.—The diagnosis of intussusception in children is based upon the sudden onset of intra-abdominal pain in a previously healthy child, accompanied by vomiting, intense tenesmus, bloody stools containing mucus, but no fecal material. There is no passage of gas. A palpable mass can be felt in the greater number of cases. Such a mass may often be felt under anesthesia, when there is apparently none present by routine physical examination. This mass is usually movable, and is not fixed as in an inflammatory swelling.

In addition to these symptoms the patient shows considerable shock and prostration, depending upon the length of time the condition has existed. Patients presenting such symptoms should be considered as suffering from invagination.

In adults, however, the picture in many instances is more obscure. When the symptoms mentioned are present, the diagnosis is easy enough. When, however, there have been a number of attacks of intra-abdominal cramping pains with periods of intermission, when at intervals the stools contain mucus and some blood to subsequently become normal in character, the diagnosis is more difficult. Complete obstruction to the passage of gas and persistent vomiting makes the diagnosis, particularly if a tumor is present.

The diagnosis of cases developing in a chronic manner may be greatly simplified by roentgenological studies made in conjunction with careful anamnesis and thorough physical examination. In some cases the actual exciting cause may not be differentiated before the case comes to operation.

Prognosis.—This is always grave, and the gravity increases directly with the length of time the condition has existed before surgical relief is obtained.

That such cases, particularly in the adult are not always fatal is

shown by the cases of Eliot and Corscaden. In 43 of 300, the necrotic intussusceptum was passed from the intestine with only six deaths. The mortality under the most favorable conditions ranges from 7.5 per cent to 30 per cent. Bolling¹ reports 51 operations upon 50 patients under twelve months of age, suffering from this affection with an operative mortality of thirty per cent. In two older children, one recovered and one died. Clubbe² records 270 cases of acute intussusception with a mortality of 20 per cent. Eighty-eight and five-tenths of these cases were under one year. In this group 100 consecutive cases show a mortality of 7 per cent.

P. L. Hipsley⁴ reports 51 cases with 4 deaths, a mortality of 8 per cent.

Treatment.—It is extremely doubtful if inversion of the patient and the injection of large amount of fluid or of air into the rectum with the hope of restoring the intussusceptum is ever justified when competent surgical help can be obtained promptly.

Immediately upon making the diagnosis of intussusception, the patient should have an abdominal section, and the sooner this is performed the better the chance for recovery.

When the abdomen is opened we have found that the least dangerous method of procedure is the one which pulls and traumatizes the intestine the least. Prompt decision, quick surgery with safe suturing are essential. A very few cases will be relieved by traction, but only within the first hours after the accident. When the surgeon determines that a resection will be necessary, it should be done without wasting time in vain efforts to replace the invagination.

An incision along the outer layer of the mesentery, particularly that of the ascending colon, will mobilize it and permit of much more rapid work. When the entire loop containing the intussusception is to be excised, doubly clamping the vessels and severing them will permit of very rapid excision.

We have found it advisable in a few cases to open the receiving intestine and draw out the intussusceptum from within, completing the anastomosis at the site of the amputation. This entails more danger of soiling, but when speed is the desideratum, it may be employed. The method of rapid and complete excision is the most satisfactory.

In the small bowel, end-to-end suture is the quickest and most satisfactory procedure. In joining the ileum to the colon, an end-to-side anastomosis may be made with the open end of the colon sutured, or a closure of each open end and a later anastomosis may be made.

Murphy buttons are not generally employed at present.

In the suturing, hemostasis must be complete, the sutures applied so that there will be no leak, but not tight enough to cause sloughing of the intestinal wall.

A very important point is to carefully provide sufficient vascular supply to nourish the bowel which remains, particularly at the line of suture.

In some of the extreme cases where the shock is marked, a complete anastomosis may sometimes not be possible. Under such circumstances the mesentery may be clamped or tied off, the intestine double clamped and excised, and the operation completed by bringing both ends of the bowel out of the wound. A rubber tube can then be sutured into the proximal end, while the clamp remains on the lower segment, to be removed later. The abdominal wound is closed quickly, and the whole operation consumes only a few minutes.

There is always a toxic fluid in the canal above an obstruction, and either on the table or as soon as practicable gastric lavage should be employed. In some cases it may be advisable to empty the distended proximal loop before the anastomosis is complete.

The patient is put to bed, kept warm and should take one pint of normal saline with adrenalin solution, fifteen minims for adults by the Fowler-Murphy method and proportionate doses for children.

Prompt decision, rapid surgery, with careful attention to detail, but no unnecessary manipulations, will bring the best results.

Chronic Intussusception.—The chronic types occur almost entirely in the adult. They are in a large number of cases due to the presence of a neoplasm, to Meckel's diverticulum, or to a previously existing acute intussusception. In one of the author's own cases, the invagination included a portion of the ileum, the caput coli, the appendix and all of the ascending colon. From the history, it was evident that the condition had existed for a long period of time, perhaps fifteen or more years. The ileum had sloughed off about 2 inches from the ileocecal valve, leaving a patent canal open at each end, lined by peritoneum, situated on the invaginated intestinal mass. The outer covering of this canal was mucosa. This patient's discomfort consisted of persistent constipation. In other instances Meckel's diverticulum is a very frequent cause. Occasionally worms act as a causative factor. Eliot and Corscaden record three such cases. In one of them the segment of bowel became necrotic, and was discharged with the tapeworm still attached. Two other cases in their series were the result of round worms.

Traumatism has been noted as the cause in a few instances. Straining and coughing may also excite its occurrence. Dysentery and colitis

both have appeared to be the only responsible conditions. Foreign bodies in the intestine have in a few instances caused its development. Ulceration from dysentery, tuberculosis, typhoid fever, and from simple ulcers of stercoraceous origin are recorded as causes.

Diagnosis.—The diagnosis of the cases developing in a chronic manner may be greatly simplified by roentgenologic studies, made in conjunction with careful anamnesis and thorough physical examinations.

Treatment.—While not so urgent in many cases as the acute forms, operative intervention is just as surely indicated. The operation may be

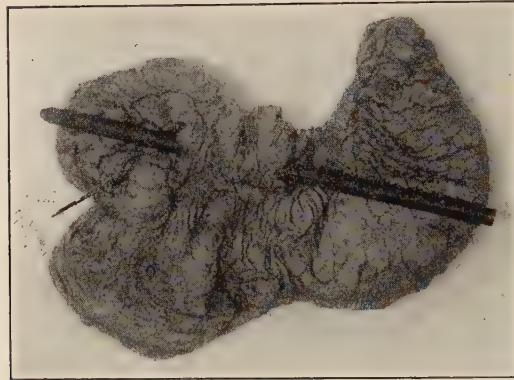


FIG. 50.—INTUSSUSCEPTUM REMOVED BY WRITER 15 YEARS AFTER OCCURRENCE.

Pointer placed within lumen of remaining portion of ileum. (Case reported in *Transactions of the Southern Surgical Association*, 1922, 276.)

planned more thoroughly and conducted with less haste than in the acute cases. It should be carried out along similar lines as in those done in emergency.

THROMBOSIS OF THE MESENTERY

Occlusion of the vessels of the mesentery whether from plugging of one of the large arteries or from a venous thrombosis is a very grave occurrence. It is by no means frequent, Ross⁷ reporting from the records of the Lankenau Hospital in Philadelphia in 30,000 surgical operations only 2 were for mesenteric thrombosis. Mitchell⁶ states that 500 cases have been recorded.

Etiology.—The factors leading to such thrombosis are changes in the rate of flow in the vessels, damage to their endothelial coats, blood stream infection, endarteritis, emboli, purpura, and phlebitis.

Change in the rate of flow alone in the normal vessels may result in coagulation of the blood during life, but is of rare occurrence except

as a terminal condition. In connection with infective processes within the blood stream or of endarteritis it becomes very important in the production of this condition.

The most prominent causative process is pathogenic or infectious bacteria in the blood stream. This may present as a low grade infection which is essentially chronic in character, resulting in endarteritis or endocarditis, indirectly producing coagulation in the vessels by embolic lodgment.

It may also occur in the form of acute blood stream infection which profoundly disturbs the metabolic processes, lowers the circulation, causes damage to the vascular endothelium, and results directly in embolism and thrombosis.

It may also result from local injury to the walls of the arteries of the mesentery, as the result of direct traumatism of minor nature during an abdominal section. Damage to the vessel by a prolonged strangulation by a band, hernial ring or intussusception, or a volvulus may be responsible for its occurrence after the local condition has been passed upon as safe.

The older authors placed rheumatism and marasmus as causative factors of this occurrence. It is now known, however, that so-called rheumatic conditions are the result of focal infections in which the bacteria or sometimes their toxins enter the blood stream. Because of these facts and an increasing knowledge of such pathology, blood stream infection is given its proper relation to this affection.

Purpura hæmorrhagica formerly considered as a rheumatic condition is now classed as due to a blood stream infection. The occurrence of mesenteric thrombosis is not infrequent in purpura. Mesenteric hemorrhage likewise occurs and may subsequently determine the development of mesenteric thrombosis. Infective thrombophlebitis of the mesenteric radicles may produce thrombosis of the entire mesentery.

Hemorrhoidal inflammation may excite coagulation in the veins which may spread into the veins of the mesentery. The author has personally observed mesenteric thrombosis attributable to a grip infection. Embolism is due to the lodgment of some clot in one of the larger arteries of the mesentery, and may be the result of endocarditis or of endarteritis. It may also follow thrombosis of the mesenteric or that occurring in other veins.

Pathology.—The study of the pathology of this affection is a very interesting one. It includes the basic principles of surgical pathology, including bacteriology and almost the entire range of inflammatory processes.

It is only within the past few years, due to the work of Rosenow and others in the laboratory, and the clinical investigations of Billings and others, that a fair knowledge of the far reaching effect of focal infections has been obtained. A very few remarks on this subject may not be amiss.

In individuals who are apparently well during adult life there are often present minute bacterial processes localized about the teeth, in the tonsil, the accessory nasal sinuses, in the prostate, in the fallopian tubes, the vulvovaginal glands, the gall-bladder or the appendix, which though inactive are none the less important to the health and longevity of the patient. It is to these foci that we must look for the blood stream infection and the many processes resulting therefrom. While apparently a far cry from these processes to the development of purpura, rheumatism, and mesenteric thrombosis, these minor conditions assume the most important rôle in the occurrence of coagulation of the blood in the mesenteric vessels.

This may also be an explanation of some of the cases of thrombosis occurring in the veins of the lower extremity after abdominal operation upon distant parts not closely or directly related. This is partly the result of slowing of the circulation incident to operation.

Since the occurrence of thrombosis and embolism of the mesentery are fraught with such high mortality, it is well to go for its prevention directly to the source of the infection.

The changes which take place within a vein as the result of phlebitis consist in damage to the endothelium, releasing a ferment inactive so long as this coat is not damaged, and the formation of a clot. This clot when due to trauma and not infected may undergo complete organization. When infected it undergoes softening, one or more fragments break away, floating in the blood stream, to find lodgment in the different parts of the body. One of such clots (embolus) may reach and plug the superior or inferior mesenteric artery or one of the large branches.

On the other hand beginning from the terminal radicles in the intestine, or one of its appendages, the clot may form there and by accretion soon occlude the entire venous plexus of a segment or all of the mesentery.

Symptoms.—Both these conditions present symptoms so alike that differentiation cannot be made between them clinically in most instances. Like sudden obstruction in the extremities the development of thrombosis and that of embolism of the mesentery are marked by

sudden sharp pain. This pain is probably due to a sudden deprivation of the tissues of blood, which acts promptly upon the nerves themselves. Subsequently the pain is due to bacterial infection and to abdominal distention with the coincident peritonitis. Shock is an early and prominent symptom.

There occur at once symptoms of obstruction of the intestine. If any stool is passed, blood is likely to be found in it. At times this hemorrhage may be profuse. There is less mucus present than occurs in intussusception. The abdomen becomes distended and very tympanitic. A noticeably distended tympanitic coil may be observed early. Soon the intestine becomes parietic and the distention very pronounced, productive of cardiac and respiratory distress. Vomiting is frequent and belching is persistent. In the absence of an acute blood stream infection the temperature may be subnormal. Prostration is marked and progressive.

Diagnosis.—This is made by the symptoms given above. Owing to its relative rarity the diagnosis is made in most cases after the abdomen is opened, the previous diagnosis being intestinal obstruction. The presence of purpuric spots with the above symptoms is suggestive.

Prognosis.—The prognosis is exceedingly grave. A few cases have been saved by operation: Sprengel one, Eliot one, Mitchell one, Finney one.

Treatment.—Emphasis must be placed on careful attention to focal infections, for by their removal this condition will occur but exceptionally. Patients with blood stream infection must be placed at rest and have some form of intravenous germicidal therapy.

The transfusion of blood has been employed in so-called idiopathic purpura. There seem to be cogent reasons why transfusion of blood in the presence of a blood stream infection should be performed with the greatest of caution. In many such cases the reparative powers have all the tax they are capable of bearing, and the addition of foreign blood entails an exaggeration of the metabolic processes within the vessels which may throw the balance against recovery. This contention is not entirely theoretical, since such unfavorable results have been observed.

The treatment proper of this condition demands immediate and radical surgical measures: in most cases a resection of the segment. In view of the recent work in blood-vessel surgery in embolism of the vessels of the extremity, the possibility of a successful embolectomy of the mesentery may be considered. In the few cases recovering, a resection was done with restoration of the intestinal lumen.

VISCEROPTOSIS

This term is applied to displacement of the organs within the abdomen from their usual or normal position. The subject received a considerable amount of attention some years ago. Some observers became so enthusiastic that they concluded the larger portion of human ills depended upon malposition of abdominal organs and that by a sort of hitching operation all these ills could be cured. This was particularly applicable to the kidney and to the stomach.

Some observers attributed all sorts of nervous and even mental disorders to the movable or ptotic kidney. Apparently they overlooked entirely the clinical fact that many patients carry kidneys showing extreme motility without giving rise to any symptoms at all, while others with but slight motion produce some very troublesome symptoms.

Ptosis of the stomach also was at this time looked upon as the source of much of the digestive disturbance complained of by individuals of this type. It is now believed that only a small proportion of ptotic stomachs, so-called, result in digestive impairment purely as the result of the ptosis.

We do not wish to be misunderstood as failing to recognize lesions of this character which are actually pathological and interfering with the life and comfort of the individual. We believe that after the first wave of enthusiasm passed and the rebound to the opposite view that professional opinion put a proper valuation upon the morbid activity of such change in anatomical relationships.

For years we have recognized that there has been a misapprehension upon the part of many as to what constituted the normal location of the abdominal viscera. This incorrect view was the result of the teaching of the older anatomists whose only knowledge of the position of the viscera was based upon observations made in the dead house with the subject in recumbency. We recognized from a wide experience as a student and teacher of anatomy that the early observations were incorrect and that most of the abdominal viscera have a considerable range of motion.

Among the chief exceptions to this are the duodenum and the attached portion of the sigmoid. The pancreas also has a more or less fixed position. These organs are fixed in position because of the close relationship to vascular trunks or to bony attachments.

Many factors play a part in determining the relationship of the abdominal organs to each other and to the parietes. Certain types of individuals of slender build with long narrow chests always show a ten-

dency to carry most of their organs low in the abdomen. In such persons the space under the rib margins is not sufficient to carry the organs that are found there in other types of build. The liver extends some



FIG. 51.—FAILURE OF ROTATION. (Fugate and Enfield.)

distance below the rib margins. Perhaps the right extremity may reach almost to the anterior superior spine of the ilium. The stomach, too, lies far below the usual site, sometimes resting with the greater curvature at the pelvic brim, in the erect posture. The transverse colon also

may lie several inches below the normal or rather generally accepted normal location.

In rare instances in such individuals in which the hepatic flexures are fixed in the usual position, particularly if these persons have a con-



FIG. 52.—NORMAL COLON. APPENDIX SHOWS ENTEROLITH. (Fugate and Enfield.)

stipated habit, the transverse colon may dip down into the pelvis, the two limbs of the loop lying side by side like a double barreled gun. This arrangement results from a redundancy in the wall of the colon produced in part by the pull of an overload. Where such an overload

has been carried for a long time and a similar retention is present in the caput and ascending colon these structures may also sag. Under these circumstances and also where there are adhesions fixing these organs in their abnormal relationship, these displacements become path-



FIG. 53.—EXTREME CASE OF PTOSIS OF TRANSVERSE COLON. (Fugate and Enfield.)

ological. Within the past few years as the result of extensive roentgenological studies of these structures these opinions derived from clinical experience have been confirmed.

The broad-chested, robust individual who has a large chest expan-

sion which enables him to cause wide excursions of the diaphragm and whose muscular planes are strong shows none of this tendency to ptosis. The reasons for this fact lie in the vigor of the individual, the muscular tone of the abdominal wall which prevents sagging and gives support to the viscera. There is considerable space beneath the dome of the diaphragm which permits a large portion of the mobile viscera to be drawn into this space on inspiration. Hence there is little tendency to sagging anywhere.

It seems reasonable to state that visceroptosis is more due to a loss of general bodily tone than that the presence of ptosis produces the loss of tone. Very careful observers have found that patients presenting for observation with visceral displacements and complaining with malaise, weakness, prostration, tired feeling, and so on, improved after the application of an abdominal support. The support gave them a sense of increased strength to the abdominal wall and they immediately began to improve. Similar results followed the use of the Rose adhesive abdominal binder. Putting these patients to bed and feeding them, particularly those that have lost flesh, improves them.

The remarkable fact is that all such patients who are treated by bandages and feel improved show no change whatever in the position of the viscera when examined by x-ray. In other words the large part of the benefit is mental. Some of it is due to the feeling of support to the abdominal wall. Almost every physician knows that as long as a person has not had his attention called to his heart it never disturbs him even when pathology is present. But if some physician calls his attention to some trifling functional disorder, he magnifies the condition greatly. The same is true of visceral conditions. When a patient first learns she has a kidney she begins to make herself miserable and possibly becomes an invalid. Physicians should be very guarded in their statements to persons with ptosis. These people are subnormal physically and easily impressed by mental suggestion. They learn to love the pose of invalidism.

What is normal in the position of the abdominal organs?

This is only relative, since what is normal for one type of individual is not normal for another. One type of individual is well built, well nourished, and vigorous; the organs show good tone and their position varies within small limits with change of position or attitude. In the other extreme, in which the individual is underfed, more or less attenuated and lacking in tone, the amount of displacement is extreme. A correct study of one hundred individuals of each type would show marked adherence to rule of type. The variations between individuals

of the same type would be infrequent and therefore to be considered abnormal. Between these groups the observation would determine the average. Neither group will show much change in the positions of the viscera except as induced by proper diet, rest, and exercise.

Operative measures in such cases, while occasionally successful, are in most cases unsatisfactory and in many the last state is worse than the first, for real and actual pathology is substituted for potential pathology.

There are, however, certain persons who develop, because of inflammatory disease within the abdomen as the result of trauma or from other reasons, adhesions fixing the mobile viscera in a ptotic position, thus materially interfering with their normal function. If this condition persists there will arise sufficient symptoms to demand relief. Surgery offers something for such patients, since there is actual pathology present which may be readily relieved and the results are usually good. Sometimes they are brilliant.

The object of the attendant, then, is to separate those cases which offer some chance of relief surgically from those in whom surgery can do little but may be hurtful. The frequency in the incidence of this condition depends upon the personal equation of the observer to a very great degree, as has been stated before.

One of the most recent contributions upon this phase of the subject is that of Coffey. (*Gastro-Enteroptosis* of this series.)

In 1906 observations on 290 unselected postmortem sections of all ages and both sexes, including a few from the fetal and senile groups, Bryant found that visceroptosis is not progressive with age.

Although the percentage of ptosis in some viscera increases with age, decreasing frequency in other viscera counteracts it in the total number. Bryant reports as the result of these observations: In males the extreme degree of ptosis was observed in 10.1 per cent of the fetal group, 12.4 per cent of the group under 40, 8.2 per cent in the group over 40, 10.2 per cent in the senile group. In the female subjects visceroptosis was extreme in 17.1 per cent of fetal group, 20.0 per cent of group under 40, 19 per cent of group over 40, 23.6 per cent of senile group.

His figures show a slight tendency to progression in females with advancing years. Forty-eight per cent of all cases showed ptosis of one or more organs to some extent.

Visceroptosis of the liver, stomach, kidney, and pylorus was not observed in the fetal group. This suggests the probability that ptosis of these organs is acquired.

Ptosis of the large intestine was frequent in the fetus and is evidently largely a congenital or developmental condition in both sexes.

Great discrepancy between the sexes in ptosis was found at the ileocecal valve, occurring in 12.1 per cent in males and in 39.4 per cent in females.

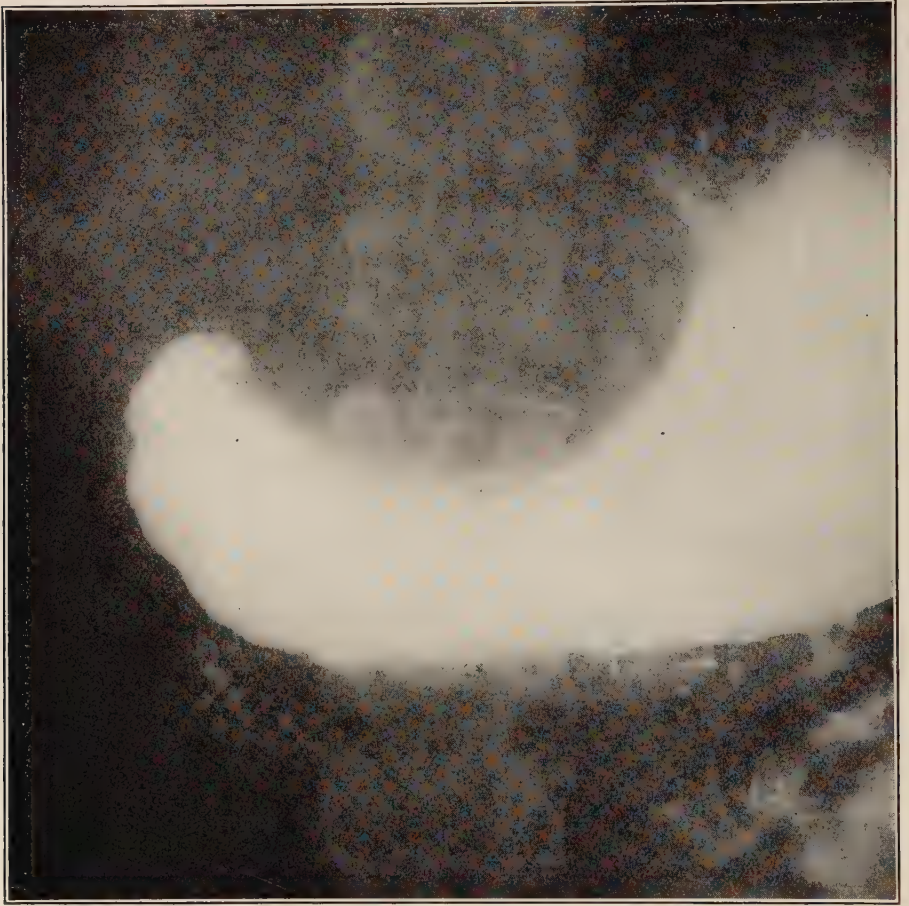


FIG. 54.—NORMAL STOMACH, RECUMBENT. (Fugate and Enfield.)

In the study of visceral ptosis if one bears in mind the facts previously presented which tend to show that the variations within normal limits are quite considerable, he is then able to recognize when such changes in position become pathological.

The simple fact that the stomach reaches down into the pelvis, while emptying itself in the usual length of time and carrying on its digestive function properly, does not signify that the position is pathological. If,

however, the stomach is a few inches below what is usual in position and does not empty itself within reasonable time or fails in its function of digestion it does become pathological.



FIG. 55.—NORMAL STOMACH, ERECT. (Fugate and Enfield.)

Again, where there is loss of muscular power or atony of its walls so that food retention is marked with resulting fermentation, then the visceroptosis itself is pathological.

The clinician, recognizing the facts as stated above and able to outline by methods of physical examination or by radiologic studies in con-

nection with the physical methods, is enabled to estimate properly the importance of the location of an organ as causative of symptoms.

When adhesions cause the ptosis or fix the organ after it has become

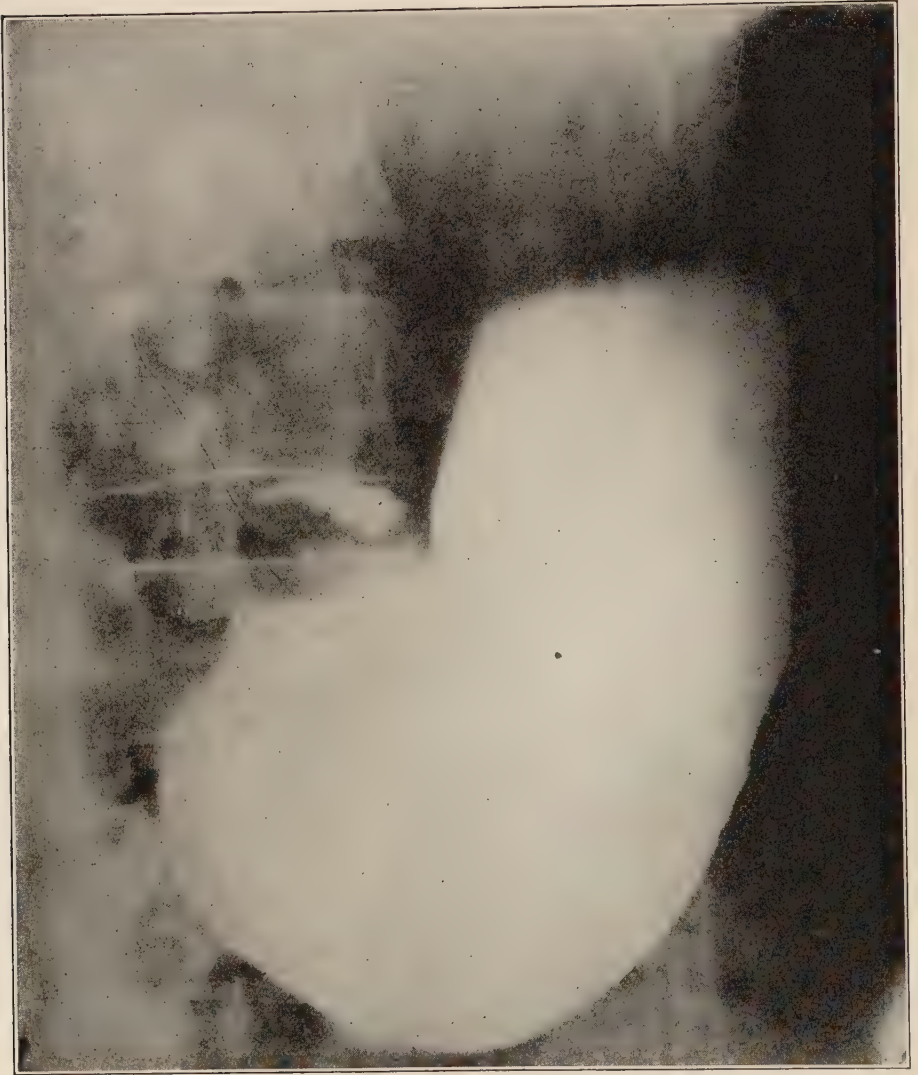


FIG. 56.—GASTROPTOSIS. GREATER CURVATURE AT THE SACRO-ILIAC SYNCHONDROSIS.
(Fugate and Enfield.)

displaced, there is present an added condition which increases the individual's complaint.

We recognize that the estimation of what amount of displacement constitutes ptosis depends largely upon the personal equation of the

observer. The bare fact of recognizing that the stomach, liver, transverse colon, or kidney is displaced does not necessarily mean that it is causative of symptoms in a given case. There must be other and more cogent reasons for instituting treatment and particularly operative treatment. It must be remembered that these individuals are subnormal, neurotic, and easily impressed and when surgical treatment fails of relief they become morbid, depressed, and frequently mentally unbalanced.

With the above views fully understood we may safely enter upon the study of the factors entering into the production of ptotic displacement.

A number of contributions appeared years before Glénard's work in 1885, notably those of Virchow and Leube. It rested with Glénard to attract the attention of the profession to the symptom complex which has since been known as Glénard's disease. The chief factors of this symptom complex are displacement of the stomach, mobility of the kidney, laxity of the colon, various digestive disturbances and usually very typical nervous phenomena. The latter are classed under the term neurasthenia.

Undoubtedly some of the nervous symptoms shown by this class of patients result from the ptosis but many of them develop as the result of nervous instability and general lack of tone.

Etiology.—The causative factors are very numerous and many theories have been proposed to cover the causation of such displacements.

No one of these alone will explain all cases.

Corsets, tight waist bands, heavy skirts play a rôle in the production of certain forms of ptosis. Undoubtedly some cases occur from general lack of muscular tone. This in turn may be in part due to a lack of nervous tone. These individuals really belong to the class of defectives. Consanguineous marriages, poverty, unhygienic surroundings, malnutrition in the parents produce such offspring.

Too little attention is paid to the nutrition of children. Improper diet, close crowding, badly ventilated houses, lack of sunshine and fresh air and improperly regulated exercise produce puny children and imperfect adults.

There is nothing which will keep the abdominal viscera in that position in which they function best as will a well developed muscular abdominal parietes. Per contra, infantile paralysis, rachitis, and poor nourishment tend to lessen this muscular tone and incidentally to cause ptosis. Premature children and those suffering from tuberculosis or

scrofula (so-called) in infancy never can keep up, either in work or play, with their vigorous companions.

Rapid emaciation after a prostrating illness in adult life acts in a similar way in its production. Frequent and promptly repeated pregnancies tend to relax the abdominal walls.

Undoubtedly perineal tears and lacerations of the pelvic floor or loss of muscular tone in these structures tend to permit sagging of the viscera. Organs cannot become ptotic when the mesenteric attachments are not abnormally loose or their vessels unduly long.

Once developed as a visceral ptosis the vicious circle forms and there is progressive increase in the displacement.

Symptoms.—The symptoms usually described as occurring in these cases are many and varied. The most frequent are lassitude, inability to stand exertion or fatigue, distress after eating, sense of weight, flatulence, constipation, nervous irritability, and cold extremities. The palms of the hands are moist and sweats easily excited. Menstrual disorders and headaches are not infrequent.

A short time in recumbency on forced feeding improves many of these symptoms, even constipation. The larger number of these individuals are long and slender with flaccid abdomens, some retraction perhaps in the upper portion of the abdomen with a pendulous contour in the lower part. It may in some cases overhang the symphysis pubis and this tends to the production of hernia.

This constitutes the *Hange-bauch* of the Germans. It indicates as a rule faulty attitude, incomplete respiratory excursion in addition to poor muscular tone. When ptosis is marked, symptoms produced by retention of noxious material in the intestines result, such as headache, poor circulation, anorexia, anemia, and marked loss of nervous control. Thyrotoxicosis may coexist.

Ptosis of the kidney excites the so-called Dietl's crisis, due to kinking of the ureter, and the pain is colicky and at times considerable. In other cases there may be no symptoms at all from renal ptosis.

It is claimed by some that the nervous symptoms from displacement of the kidney are most serious, progressing from general ill feeling to marked melancholia or even to a toxic psychosis with homicidal tendencies.

The claim is also made that by fixation of the ptotic kidney such symptoms may be fully relieved. Certainly the assumption of recumbency sometimes relieves Dietl's crisis.

When adhesions coexist with ptosis we find added to this picture all the symptoms of chronic intestinal stasis.

Pathology.—This consists for the most part of displacement of the organs under consideration, which may progress to the point of angulation. For instance after the stomach drops far into the pelvis there may occur an angulation at the pylorus which prevents prompt emptying of the stomach and causes food retention (stasis). This is more likely to occur if there are present adhesions binding down the stomach at this point.

Prolonged retention of food in the stomach may lead to overdistention of the organ and atony of its musculature. This in turn again increases the distention and the vicious circle is complete.

Ptois of the caput coli may, by sliding downward of this portion of the gut with a fixation at the ileum by Lane's kink or a Jackson veil, produce a chronic intestinal stasis, or, in some cases in which the adhesion is strong, a true ileus.

Glénard claims that ptois begins at the hepatic flexure of the colon and the other events incident to the disease follow. It is quite possible that the ptois of the right colon is congenital. Meinert looks upon the pulling and stretching of the nerve fibers within the mesentery as important in the production of symptoms. Schwerdt considers the nervous system to be primarily at fault; that because of the toneless fiber of the individual the functions of the abdominal muscles are not normal, resulting in loss of intra-abdominal pressure, and ptois follows.

Diagnosis.—The presence of the more or less characteristic symptoms mentioned above promptly calls attention to this condition. The physical examination promptly discovers the anatomical relationship and if it does not the x-ray examination will give an accurate idea of the location of the different organs.

The most important question to be determined is to what extent the symptoms presented are dependent upon the ptois and to what degree they are due to bands and therefore removable.

Prognosis.—The prognosis is usually good notwithstanding the low state of nervous and nutritional tone. Some of these patients may be fully restored to usual health. This is more particularly true of the cases occurring in youth before habits are fixed. Slouchy gait, sluggish respiration, lazy movements, lethargy may all be corrected by correct attitude, proper breathing exercises, physical culture, good food, plenty of sleep and rest, cold baths, outdoor life, and fresh air and sunshine. There is nothing like good blood to put "pep" into an individual. An undernourished person cannot be buoyant with energy. The army discipline is remarkable for the improvement of the physique of a soldier. If there were no other reason for universal military training the difference

in the physique, the mental vigor, the attitude, and the alertness between the rookery and the trained soldier is sufficient to justify this policy.

Gastroptosis is probably one of the most frequent forms of this affection. It may occur alone or in conjunction with general visceroptosis. It occurs more often in women than in men. It assumes importance in direct proportion to the failure of the stomach to propel the food through the pylorus within a reasonable time.

This failure may be due to pyloric obstruction, from spasm, from stenosis due to ulcer or malignant disease or because of adhesions. It may be due to atony of the gastric musculature from prolonged overdistention. It may also be the result of loss of nervous supply to the gastric muscularis.

This condition is progressive since the prolonged overdistention exaggerates the atony, so that relief is not obtainable by the passage of the contents through the pylorus or by regurgitation or vomiting.

In the early stages of this condition much may be accomplished in the way of treatment by gastric lavage at frequent intervals. The use of hot water carrying small amounts of chlorid of sodium and bicarbonate of sodium will aid in the restoration of the muscular tone and also in the stimulation of the gastric secretion. It also takes off the excess weight carried by this organ and this in turn permits of its contraction in size. Peristalsis becomes more active. The use of *nux vomica* or its alkaloid will favor increased power of contraction. Minute doses of pituitrin may produce the same result.

When such measures fail of relief after a fair trial, the question of surgical intervention comes up for consideration.

A number of methods of operation have been recommended to overcome this affection. Most of these depend upon the employment of the already attenuated gastric peritoneal folds to act as support for the stomach. Others depend upon the production of a sling for the stomach by attaching the mesocolon and the transverse colon to the abdominal wall. In this operation, proposed by Coffey, the stomach after the operation was supposed to rest on this shelf formed of the omentum and colon at their attachment to the abdominal wall (Coffey).

Beyea's operation consisted in shortening the lesser omentum by plicating the structure by a number of interrupted catgut sutures.

In other cases in which an abdominal operation has been performed or where a pelvic infection has occurred where the omentum has become adherent to the tissues within the pelvis, thus exerting a tug on the colon and stomach, the condition is pathological.

The two previous conditions do not need surgical treatment, nor will

such measures prove beneficial. In the latter group of cases, however, the correction of the pathological conditions causing the ptosis, thereby relieving the stasis, may prove very beneficial.

The operative steps employed consist in the separation of all adhesions, suturing over all raw surfaces and prompt closure without drainage. In rare instances only, where from long duration of the pathology and because of atony of the colon, or in case of megacolon, the isolation of a portion of the intestine or colectomy may be appropriate.

Further consideration of the effects of ptosis and the results of interference with fecal flow will be found under Intestinal Stasis.

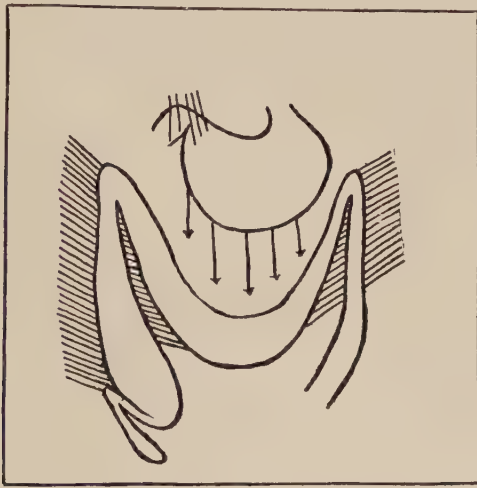


FIG. 57.—SHOWING PTOTIC DISPLACEMENT AND RESULTING TUG. DIAGRAMMATIC.
(After Lane.)

These operations have not appealed to the surgical profession sufficiently to receive general adoption.

Occasionally they produce such improvement that the results seem brilliant. The end results of such treatment in the large majority of cases are not satisfactory. The reason for this seems apparent. In the effort to relieve a functional disorder, real pathology is produced to replace potential pathology. The mobile ptotic stomach is made the more or less fixed stomach which, because of a diminution in its motility, loses even to a greater degree its tonus. The adhesions formed in such conditions lessen its contractile power and interfere with its regular rhythm so that there is marked interference with its normal function.

Ptosis of the Colon.—This condition may develop alone or in conjunction with ptosis of other viscera, either stomach, liver, or kidney.



FIG. 58.—PTOSIS OF THE COLON.

Note shepherd's crook appendix. Chronic stasis. Mild type.

In the latter instance it is only part of a general insufficiency of muscular and nutritional tone (Figs. 57, 58).

In other instances the transverse colon passes obliquely across the abdomen from the right iliosacral junction to the splenic flexure. No ascending colon or hepatic flexure is observed. This is a congenital condition due to a developmental error or failure to complete the develop-

mental process. Rotation of the intestine may be incomplete. Such a condition while unusual and somewhat infrequent is perhaps not harmful and perhaps requires no treatment.

Nephroptosis.—Unusual mobility of the kidney is observed with considerable frequency. Increased mobility occurs about ten times as frequently in women as in men. It is noticed more often upon the right side than upon the left. This greater mobility of the right kidney is supposed to be due to a longer vascular pedicle upon the right side than upon the left. This seems doubtful since the vein from the left kidney crosses the spine. It would appear that the vessels have a freer sweep from their point of attachment on the right side and that the defect which permits motility lies in the fascia.

Three degrees of abnormal mobility are usually recognized. The first permits of palpation of the organ. The right kidney is palpable in most healthy individuals. The left kidney is more difficult to palpate. The second degree is evidenced by the ability to outline the upper pole by palpation. The third degree of mobility is shown by observing the kidney in wide excursions throughout the abdomen. The kidney in the latter case has escaped from the fascial sleeve. Its vessels permit the wide excursion, the circumference being 3.14 times the length of its radius.

There is a marked variation in the amount of mobility shown in some of these cases. There is also remarkable variation in the symptoms resulting from this mobility. The mobile kidney may exist for long periods without the knowledge of the individual and is part of a general lack of tone. No symptoms may be elicited from the patient referable to this form of ptosis. When his attention is called to the mobility, his mental worry is immediately increased. Under certain conditions, however, even when the mobility is slight, kinking of the ureter or of the vessels results in sharp attacks of pain, marked nervousness, and sensation of weight.

The fact that a kidney is not mobile does not mean that it is not ptotic. Not infrequently a kidney may be fixed in a ptosed position. Occasionally there may be a kinking of the ureter in such type. When there is interference with the vascular supply of a mobile kidney the organ becomes increased in size. Because of its weight the amount of displacement is increased.

Causes of Mobility.—The kidney is loosely held in place and a certain amount of motion is desirable for the performance of its function.

In addition to its fibrous capsule it is surrounded by a loose fatty layer. When this is intact there seems to be little hypermotility. After

wasting diseases in which the loss of this fatty layer is rapid, the kidney is often found to be more mobile.

Conditions which lessen the tension of the peritoneum or diminish the muscular tonus of the abdomen tend to cause mobile kidney. Repeated pregnancies are prolific causes. Particularly when the woman nourishes and lifts a vigorous child there is marked tendency to develop this form of ptosis.

Symptoms.—It is very likely to be discovered by accident. The physician in examining for other conditions frequently notices it first. The patient may discover a movable mass which has caused no inconvenience and she is curious to know its significance.

In some cases the symptoms are slight. In other instances the patient may be nervous, ill-tempered, uncomfortable, unable to work without being able to assign a reason. In still others the symptoms present suddenly with nausea, vomiting, and pain. This pain is directed along the course of the ureter to the groin or testis.

Following relief from kinking of the ureter the patient passes a large amount of urine. During the attack it may have been scant.

Examination of the abdomen reveals a mobile mass which may readily be pushed into the loin. Many of these patients are extremely neurotic and there are usually evidences of ptoses of other organs.

Treatment.—Many cases require no treatment. In some cases treatment will provide no permanent benefit. The use of abdominal supports, pads, and similar plans of treatment have not proved successful.

Probably the most satisfactory treatment is recumbency for six or more weeks with forced feeding. Such treatment is employed to permit the organ to reach its normal habitat and by the increased deposit of fat it is hoped to hold it there.

Some years ago great stress was given to the operative treatment for this condition. Many believed that by fixing the kidney as nearly as possible in its normal position the symptoms would be relieved and the great variety of nervous symptoms would be relieved.

Some cases showing Dietl's crises were improved. Others were unimproved. In some instances the organ was sacrificed to get rid of the discomfort. At present the operation of nephropexy is only performed to meet some particular indication such as the relief of kinking.

The methods most in use are suspension by stripping a portion of its fibrous capsule, passing this through an opening in the lumbar fascia and suturing it there and also to the capsule again.

Rest, diet, carefully graduated exercises in many cases may avoid the necessity for operative measures.

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CHAPTER IX

DISEASES OF SPECIAL STRUCTURES

THE RETROPERITONEAL GLANDS

The lymphatic structures in the retroperitoneal space are of great significance in protecting the organism from infections entering by way of the intestine. The greater number of such infections are properly overcome by the lymphatic glands. In a certain proportion of cases, however, the dosage of bacteria is too large to be promptly and successfully overcome.

Following the immediate contest, the bacteria remain localized for a more or less extended period of time during which combat between the reparative forces of the individual and the bacteria continues. The resistance of the body is built up to its highest protective efficiency in this period. This is well shown in tuberculosis of these glands.

The affections which are most likely to involve the retroperitoneal lymph glands are tuberculosis, pyogenic infections, Hodgkin's disease, lues, typhoid fever, pneumococcus infection, malignant growths, particularly sarcoma, both primary and secondary.

Surgically the most important of these infections are tuberculosis and sarcoma.

Tuberculosis.—The invasion of these glands by the bacillus of Koch usually occurs during infancy and early childhood. Occasionally this results from the milk of a tuberculous mother. It may also be of bovine origin.

The nonmotile bacteria are transported slowly and because of this fact considerable immunity is developed. The result is that the infection produces a slow form inflammation which only becomes active when the general health is below par from an intercurrent infection. Occasionally the process is very acute from its inception and the child may die from miliary tuberculosis.

In cases in which the bacillus is able to multiply, the tissue reaction to its presence is characteristic. Mitosis of the cells takes place and small nodules are formed which show an increased number of lymphoid cells, epithelioid cells, and giant cells. In such tubercles the bacteria are found particularly at the periphery of the giant cells. There is marked

hyperplasia of the lymphoid tissue. The fibrous structure surrounding the gland is much thickened. In the tubercle itself no blood-vessels are found. Because of this fact there is ever present a tendency to softening and caseation of this structure.

In certain cases cicatrization and contraction occur. In others the lymph node becomes replaced by a calcified mass. After caseation occurs the process may become quiescent and the bacteria remain inactive but viable for long periods of time. In rare instances rupture of a caseous gland occurs and an acute peritoneal tuberculosis is excited.

Mixed bacterial infection may occur and the active inflammatory process destroy the tubercle bacilli. A similar acute pyogenic contamination may result in a retroperitoneal abscess which in turn may produce a suppurative peritonitis. This is of rare occurrence.

Occasionally the tuberculous process results in marked enlargement of these glands. When the tuberculous infection involves the intestine as well, large masses may be palpable through the abdominal wall.

The symptoms of this form of tuberculosis may be very slight for a considerable time. The individual is anemic, pasty and more or less below par. Occasionally there is a slight rise of temperature.

If the process becomes inactive the patient may show considerable improvement in general health. When the intestinal or peritoneal involvement is considerable the abdomen becomes distended, has a doughy feel with but little or no tenderness. Occasionally masses of considerable size may be felt.

Progressive increase in the pathology results in loss of flesh, night sweats, and a low grade of fever.

In some active cases the presence of an intractable diarrhea is observed.

In miliary tuberculosis the intestines, the lymphatic glands, the peritoneum, the lung, the pleura and perhaps the meninges are involved and the cases run a rapidly fatal course.

The diagnosis may be made only at operation for other affections in some instances. The presence in the abdomen of palpable masses of irregular shape not otherwise explained are strongly indicative of tuberculosis of these glands. The presence of a demonstrable tuberculosis of the peritoneum with ascites or a tuberculosis of the intestine with blood and mucus in the stools points strongly to this condition. A low grade of fever, anemia, complaint of fatigue on little exertion, night sweats, point to this disease.

The von Pirquet or Calmette reaction is very valuable in children in arriving at a diagnosis.

The discovery of irregular nodular masses in the posterior part of the abdomen by means of pneumoperitoneum and roentgenograms is very strong evidence of infection of the retroperitoneal glands.

Undoubtedly many cases of tuberculosis of the retroperitoneal lymph nodes occur without recognition.

In practically every recovered case of peritoneal tuberculosis there has been present an involvement of these glands in some degree. This appears to prove that recovery does occur in many cases suffering from this particular type of this disease.

Every case in which tuberculous adenitis is demonstrable clinically or upon the operating table is of serious nature. Many such cases go on to recovery. Others develop either osseous or pulmonary tuberculosis later in life. Many of these cases continue with the tuberculous process quiescent and die of some intercurrent disease.

The prognosis in cases of this type should be guarded but the outcome is not hopeless.

Treatment.—There is practically no surgical treatment for tuberculosis of the retroperitoneal glands. Much may be done surgically for concomitant and perhaps causative lesions such as tuberculous appendicitis, tuberculous salpingitis, tuberculosis of the peritoneum and occasionally tuberculosis of the intestine. Extirpation of a localized lymph adenitis and excision of a localized intestinal tuberculosis may be successfully accomplished. When, however, any of these lesions becomes advanced the case is hopeless.

The measures to be employed in cases of this local form of the affection are for the most part hygienic. Fresh air, sunlight, good, substantial, and easily digested food will do much to bring improvement to these individuals.

The question of the value of tuberculin in such a condition is not determined. Apparently the patient is doing all that is possible to build up an immunity and the treatment should be planned to aid this.

Heliotherapy probably has a considerable field here.

Iron, arsenic, and general tonics are indicated.

Hodgkin's Disease.—It is not at all infrequent in Hodgkin's disease to find the retroperitoneal glands enlarged. The retroperitoneal nodes are usually enlarged after axillary, cervical and inguinal glands are easily palpable.

These glandular enlargements are painless and are accompanied by a characteristic blood-picture.

There is marked leukocytosis, sometimes as high as two or even three hundred thousand.

The red blood cells are diminished in number, the color index is low and the hemoglobin content is diminished.

This type of pseudoleukemia is progressively fatal. A few cases have responded to the repeated and persistent use of x-ray treatment.

The use of arsenic has proved beneficial in a few instances.

Operation is contraindicated. A gland may be removed under local anesthesia for study.

Acute Suppurative Adenitis.—This affection occurs with extreme rarity. Occasionally infections in the inguinal group of glands extend to the deeper peritoneal glands. The infrequency of this occurrence may easily mislead the observer.

The symptoms are those occurring in any lymph-node enlargement, pain, tenderness, heat, and swelling. Leukocytosis is present. The process tends to point after suppuration. It is well, therefore, to bear the possibility of this occurrence in mind.

The treatment of suppuration in these glands must be conducted on general surgical principles.

Retrocecal Abscesses.—These are nearly always of appendiceal origin. Occasionally, however, they develop from tuberculosis of the vertebræ or from a localized tuberculosis in the cellular planes. Disease of the ileum or sacro-iliac joint may produce such an abscess.

Extraperitoneal gunshot wound of the colon or cecum may excite suppuration or an acute phlegmon. I have seen such abscess follow an ulcerative perforation from malignant disease of the caput coli.

The symptoms of such abscesses depend upon the causative factor. Cold abscesses progress very slowly, while acute processes run a very rapid course. There is induration, a hard, brawny feel in the lower right quadrant. Usually this is unaccompanied by vomiting. Some pain and marked tenderness is observed. Leukocytosis is usual.

If bulging occurs toward the abdominal cavity, the symptoms of localized peritonitis will be present. The rigidity will extend throughout the abdomen perhaps. The brawny hardness, the tenderness, occasionally redness without vomiting or distention in the early part of the affection make the diagnosis. The greater part of the soreness and pain is posterior.

These are serious complications and should be treated promptly by free incision and drainage through the loin.

Localized Abscess.—These abscesses form either as extra- or intraperitoneal collections.

In cases convalescing from acute general or from localized peritonitis the formation of pockets of pus is not infrequent.

Extraperitoneal accumulations are found in the pelvirectal space, in either broad ligament, in the retrocecal space, in the perirenal fossa and under the diaphragm.

Such purulent collections may be a sequel of an attack of peritonitis or may develop from local infective processes and become causative of peritonitis.

Those abscesses which are strictly extraperitoneal lie outside the parietal peritoneum. Frequently such lesions are so closely related to intraperitoneal structures, in many instances following an intraperitoneal infection as appendicitis, cholecystitis, suppurative hepatitis, etc., that they are to all intents and purposes peritoneal lesions. In some cases one or more localized intraperitoneal abscesses are found among the coils of intestine. The development of such a lesion adds much to the gravity of an attack of peritonitis.

Symptoms.—Following upon a period of improvement after an attack of peritonitis or subsequent to a few days of apparent improvement after operation the patient develops a chill or chilly sensation. This is followed by a high grade of fever, sometimes 103 to 104 degrees. The pulse is increased in frequency. Pain, tenderness, soreness, and rigidity are the local evidences of such sacculation of pus.

The exact location of the induration and the pain will usually be over the abscess. This is not invariable, however. Dulness on percussion will be elicited if the abscess is approaching the surface and is not covered with coils of intestine.

The diagnosis of a localized inflammatory process may be made earlier than the exact location of the pus can be determined.

The localization of such pus pockets depends largely upon the causative lesion. The history of the case will usually point to the source of infection and assist in the localization.

Treatment.—The treatment of suppurative lesions of this character depends upon several factors. Among these may be mentioned the causative lesion, since this often determines the preoperative management and the direction of surgical approach when the abscess is to be opened. The location of the pus pocket and its tendency to point in a certain direction also determines to some extent its management.

The presence or absence of a blood stream infection has a most important bearing upon the treatment.

It is generally recognized that some additional danger is assumed when extraperitoneal purulent exudates are opened through the peritoneal tissues. Notwithstanding this many surgeons have long recognized the importance of dealing transperitoneally with the causative lesion in

many of these cases, particularly those due to appendicitis, cholecystitis, ruptured gastric ulcer, and pancreatitis.

The final outcome will be more satisfactory when this radical method is employed in preference to a timid and small extraperitoneal drainage. The reason for this lies in the fact that removal of the causative lesion checks further bacterial invasion from this source. When this is accompanied by a free drainage the best results will be obtained.

There are certain cases, however, which will need a different treatment. Those cases in which localization is slow and the symptoms do not definitely indicate a point of attack should be treated by local application of cold with general measures to aid the flagging vital forces. In some instances cold causes great pain, and if so, heat may be substituted.

A few abscesses in the pelvis or elsewhere which are evidently extraperitoneal may best be handled by extraperitoneal incision and drainage. This is particularly true of perirenal infections and abscesses pointing in the vaginal vault.

This may be only a palliative measure to carry the patient through a condition in which operation is dangerous. Subsequently more radical surgery may be undertaken with increased safety.

(For other and collateral conditions the reader is referred to other monographs in this series.)

ASCITES (HYDROPS OF THE PERITONEUM)

In its widest sense this would mean the presence of any free fluid within the peritoneal cavity. The extravasations occurring during an acute inflammatory process do not accumulate in any considerable quantity and they subside promptly. The term ascites is, therefore, applied to the slow forms of fluid collections within the abdominal cavity when they tend to persist for some time.

The examples of such an accumulation are dropsical effusions occurring in the course of general anasarca from renal or cardiac disease, from cirrhosis of the liver, from malignant disease of the peritoneum or some of the viscera, particularly the liver, from Banti's disease, and from chronic peritonitis, usually in the form of tuberculosis of the peritoneum.

The fluid found in the latter groups differs materially from that occurring in the acute inflammatory processes. This exudate differs from ascitic transudates in the higher specific gravity, 1.015 to 1.030, and in the greater number of cells present. The higher specific gravity is

almost wholly due to the higher percentage of albumin present in exudates, 4 to 6 per cent. Hydropic accumulations show a specific gravity of 1.005 to 1.015, and about one to $2\frac{1}{2}$ per cent of albumin.

In addition to the causative conditions mentioned above as complicated by hydrops there is a distinct form known as chylous ascites. In this condition the fluid presents a milky character, otherwise is quite like that of any other transudate. This type will be discussed later.

Etiology.—The causes of peritoneal dropsy are cirrhosis of the liver, particularly the atrophic variety, cardiac disease, disease of the kidneys, carcinoma, Banti's disease, leukemia and splenic anemia, damage to the lymphatic trunks, filariasis, tuberculosis of the peritoneum. Emphysema by crippling the heart and pulmonary circulation may cause it. Lues also may cause its development. The same is true of sarcoma or retroperitoneal cysts. Thrombosis of the portal vein may also cause dropsy. Abscess of the liver, echinococcus cysts, cysts of the pancreas, cysts of the mesentery, tumors of the stomach, enlarged retroperitoneal glands and diseases of the thoracic duct, tumors within the mediastinum, and aneurysm may also cause it.

The mechanism of the retention of fluid in the cavity probably is simpler than would appear on first consideration. The explanation of the inability of the tissues to retain fluid within the vessels in cardiac or renal disease seems to lie in the back pressure from flagging heart and the increased peripheral capillary resistance. This produces a hydropemia in which the tissue cells are water-logged and have lost that normal balance which enables them to keep the water within certain limits. Dr. Martin Fischer, of Cincinnati, has developed a very ingenious theory to explain this ability to keep a balance between the albuminous or colloid substances and the water in the tissues. There is another factor of importance in cardiorenal dropsy and that is the decrease in renal efficiency as regards elimination.

In hepatic cirrhosis and the so-called nutmeg liver there is a marked mechanical obstruction to the portal circulation which does not permit the veins to return the usual amount of blood through this organ. This leads to a vascular stasis and the passage of fluid into the peritoneal space. The excess water in the blood in such cases enters the sac as a transudate. There is an effort upon the part of the organism to take care of this venous stasis by dilatation of the abdominal veins. The superficial veins become greatly dilated, forming the caput medusa in some instances. Rarely is this compensation sufficient to take care of the water and the albuminous material.

Tapping brings, as a rule, only temporary relief in either of the three

preceding conditions since the transudate accumulates very rapidly. This occurs so rapidly in some cases that one wonders where all the fluid comes from that is removed by aspiration. Very large amounts have been removed by frequent aspiration. W. Hale White⁴⁷ records a case of perihepatitis in which thirty-five taps recovered seven hundred and ninety pints of fluid. During an illness of four years twelve hundred liters of fluid were removed by forty-seven taps in Putz's³¹ case of atrophic cirrhosis.

In the beginning of ascites due to obstruction the fluid escapes because of mechanical back pressure. Later in the affection, however, there is an added factor in the permanent dilatation of the capillaries which favors the persistence of the dropsy. In certain types of the affection the changes in the capillaries and in the endothelial cells of the peritoneum play a part in the development of the hydrops. In malignant disease and peritoneal tuberculosis, as well, these factors are active. The peritoneal cells apparently lose their power of taking up and disposing of the fluid. The pressure of tumors on the vessels is also important in cancer, and in tuberculous disease the granulomata upon the peritoneal surface tend to lessen its power of absorption so that the highly albuminous fluid will not be taken up. When, however, the abdomen is opened and the albuminous material escapes, a more acute reparative process is excited and absorption is improved, some of this class of cases recover from this very simple procedure.

Symptoms.—The presence of ascitic fluid in the peritoneal sac is indicated by gradual enlargement of the abdomen. This may continue until the respiration is materially impaired. There is an accompanying edema of the extremities in some cases. The skin of the limbs pits on pressure, showing a marked amount of fluid in the cellular spaces. The skin over the abdomen becomes stretched and glazed as the dropsy increases. There may be pitting on pressure in this region also if due to anasarca. The navel is level with the skin or protrudes similar to the appearance in ovarian cyst. The navel in fibroid uterus without hydrops never protrudes unless a hernia is present. If an umbilical hernia is present the sac will contain fluid. Respiration is impaired, cardiac action is embarrassed and decompensation is present. Fever is absent except in some of the cases of tuberculous lesions.

Diagnosis.—The first consideration is to determine the presence of fluid.

On inspection during the early progress in the absence of anasarca, the abdomen will be found increased in size. In recumbency the flanks are bulging and the anterior abdominal wall is flattened in the center.

When the patient is sitting or standing there will appear a bulging in the lower portion of the abdomen. The superficial abdominal veins are evident, and as the case progresses the dilatation increases. Fluctuation is readily elicited by careful examination. One hand flat upon one side of the abdomen easily recognizes the wave imparted by a sudden quick but gentle blow upon the other side. The hand of an assistant placed perpendicular to the wall in the middle line with the inner edge down prevents the abdominal muscular impulse from confusing the observer.



FIG. 59.—LARGE OVARIAN CYSTOMA.

Note similarity to case of colloid carcinoma and to ascitic form of peritoneal tuberculosis. (Author's case.)

Percussion reveals the dulness in the flanks at the site of the fluid, the level changing with any change of position. The intestines lie on top of the fluid, hence tympanitic sounds are obtained on percussion at the center of the abdomen, when recumbent. A tympanitic note is elicited in the upper sector, dulness in the lower, when erect. These signs so elicited make the diagnosis clear from ovarian tumors.

The tympanitic note is, outside of a central dulness in ovarian cyst of considerable size, the only kind which leads to confusion (Fig. 59).

In some ovarian cysts there is evidence of free fluid in the flanks.

Such a case may be confusing. Usually in such a case a sharp deep tap on the abdominal wall will reveal the outline of the cyst wall beneath the abdominal parietes. Palpation will also facilitate its recognition. Succussion is sometimes of great value in this class of cases.

Sir Spencer Wells made a mistake in the diagnosis of an ascites, mistaking it for an ovarian cyst, and as a result the present treatment of tuberculous peritonitis originated.

Malignant tumors and multilocular ovarian cysts may be mistaken for certain tuberculous cases of ascites in which nodular masses may present.

Cysts of the ovary may usually be recognized by bimanual examination. Some large ones are not easy of differentiation.

The diagnosis of cirrhosis is based upon the history of morning nausea and perhaps vomiting. Hematemesis may have occurred. There is absence of edema in the limbs or other parts of the body. Caput medusa or dilated abdominal veins are observed. The liver dulness is diminished. There is absence of primary cardiac or renal disease. Some cases of cirrhosis are due to cardiac disease from back pressure. The patient is a drinker, shows no loss of flesh, no fever or other evidence of tuberculosis.

Tuberculosis of the peritoneum is recognized by the absence of any of the other causative conditions by the difference in the history, by a gradual loss of flesh and strength. This may not be prominent early. There is a doughy feel to the abdomen which is more prominent than in ascites of other types. There is often irregularity in the areas of dulness. If necessary (and this is not often) paracentesis for diagnostic purposes may be employed. If there is any suspicion of echinococcus cyst this method should not be used. Anesthesia sometimes makes the diagnosis easy.

Tuberculosis occurs in young persons up to middle life, forty years. There is loss of flesh and strength. The fluid changes with position. The tuberculin tests will be positive. Fever is not always but usually present.

Leukocytosis is absent unless a mixed infection occurs.

Carcinoma is slow in development, occurs after forty usually. It shows irregular nodules, some history of carcinoma elsewhere or some intestinal disturbance. There is either diarrhea or constipation. Perhaps these symptoms alternate. The patient presents loss of flesh early, and late in the disease cachexia is noted. Masses may be palpated. When dropsy presents in carcinoma the case is far advanced. Pain is not a prominent or early symptom. Obstruction to the fecal flow, either

sluggish intestinal function, partial or even complete obstruction may first call the patient's attention to the condition.

The diagnosis should be made accurately by the history of the case and by the physical findings. In case of doubt the use of roentgenologic examination is of great value. This is particularly true of beginning cancer.

The use of air insufflation as an adjuvant to the x-ray investigation is worth while. Laparoscopy may also be employed in obscure cases.

The exploratory laparotomy as urged by Urban Maes in a recent editorial in *Surgery, Gynecology and Obstetrics* is worthy of consideration. It should be employed when other methods fail to clear up the case but this is of rare occurrence.

Banti's disease is recognized by splenomegaly, enlarged liver, abdominal dropsy and by the blood-picture.

Lues of the liver may cause dropsy. This may be recognized by the clinical history and by the therapeutic test.

It must be remembered that the Wassermann test has been found negative in two-thirds of the cases involving the liver. One of my own cases with large gumma of the liver had been operated upon one year previously in another city and pronounced cancer. This aided in arriving at a diagnosis in the presence of a negative Wassermann. The mass melted away like magic under mercury rubs.

A provocative Wassermann should be made.

Prognosis.—The prognosis depends upon the causative factor. Patients suffering from cardiac and renal dropsy of the peritoneum rarely last two years. Cases of cirrhosis may continue somewhat longer. Carcinoma involving the peritoneum and producing dropsy is usually fatal within the year. Luetic dropsy may be cured.

Tuberculous ascites usually shows marked improvement after operation, some cases going to recovery. Dowd's case recovered and lived twenty-one years, when operated upon after this lapse of time for other trouble the peritoneum was free of tuberculosis and of adhesions.

Treatment.—Paracentesis has been much employed to relieve the overdistention from ascites. In cardiorenal cases this may become necessary to the comfort of the patient. It should be deferred as long as possible, dependence being placed in saline purgation, cardiac tonics to restore the cardiac compensation and the use of diuretics until tapping is imperative. Some cases of this type have been relieved entirely.

Cirrhosis should be treated by change of the mode of life, the ingestion of small amounts of fluid, and occasional paracentesis. Talma's operation of omentopexy may be tried. Perhaps more may be accom-

plished by external or internal drainage of the biliary tracts with a view to diminishing the hepatic engorgement.

Banti's disease is treated by removal of the spleen with fair degree of success. I have been able to show marked improvement in such case in which splenectomy was done with removal of gall-stones and drainage of the gall-bladder at the same sitting.

Malignant cases may be best handled in their early course by frequent physical examinations which detect them early, otherwise little can be done.

Tuberculous ascites is best treated by abdominal section with general tonic measures, fresh air, and sunshine. Light treatment is beneficial. It appears that radium has been employed with considerable benefit according to recent reports.

CHYLOUS ASCITES.—In order to understand this condition some consideration must be given to the formation of the chyle.

This fluid, which is found in the lacteals after a meal, is an opaque, milky white fluid, neutral or alkaline in reaction.

It is very like the lymph in consistence, reaction, and specific gravity. It has, in addition to the usual ingredients found in lymph, a considerable percentage of oily or fatty material, which has been emulsified by digestion. This oily or fatty material appears under the microscope as exceedingly minute globules of fat surrounded by an albuminous coating. The globules are nearly uniform in size, measuring about $1/3000$ of an inch (0.3 m.). The number of these globules depend upon the amount of fat in the food. This fatty material is responsible for the opacity and for the whitish color of the chyle. That these globules are formed of the fat has been definitely proved by their solubility in ether. That the covering about the particles of fat is albuminous material is proved by the fact that the addition of water or acetic acid to the chyle dissolves the albuminous material, and oil globules appear. It is a well-known pharmaceutical fact that emulsions of fat are readily made by mixing it with the albuminous substance of an egg. Undoubtedly a similar emulsion is made in the intestine preparatory to the absorption of the ingested fat.

The exact method by which this fatty substance enters the lacteals is not entirely clear, but the consensus of opinion seems to be that the globular cells covering the villi seem to have the property of aiding the passage of fatty substances into the lacteals.

After entering the lacteals, the chyle passes upward to the receptaculum chyli; thence through the thoracic duct to the subclavian vein. Chyle taken from the villi or from the lacteals near them contains no

solid or organized bodies, the fatty molecules being the only substances floating in the stream. This fluid does not coagulate spontaneously. As it moves toward the thoracic duct, and particularly after traversing one or more mesenteric glands, the oil molecules lessen in number and cells resembling leukocytes, called chyle corpuscles, appear, giving it the property of coagulation. The number of these corpuscles and the coagulability increase as it nears the opening of the thoracic duct.

The clot of chyle is similar to a blood clot with the red cells left out. It is always softer, more pliable and moister than blood clot. A quantity equal to one-sixteenth of the body weight of an animal passes through the thoracic duct in twenty-four hours. The amount of lymph passing into the thoracic duct in twenty-four hours has been estimated as about two-thirds the amount of blood in the body. The passage of chyle into the blood is one of the very important processes in nutrition. A study of the changes occurring in the blood stream in the disposal leads us too far into the subject of metabolism.

The above study of the transportation of the chylous fluid is important, however, in consideration of the rare condition of chylous ascites.

This affection is not of frequent occurrence, but is never a physiologic process, and the fact that chyle or a milky fluid is present in the peritoneal sac always means some pathological change permitting its passage from the lacteal radicles or from the receptaculum chyli or through the lymph glands and through the endothelium to appear visible to the naked eye. Often the pathology causing this condition is not apparent and may be quite obscure. The writer has observed the presence of chylous fluid escaping from the peritoneal cavity during an operation for hernia in an otherwise apparently healthy individual. The health of such an individual often has apparently not been affected by the presence of the chyle or by the causative factor in its escape from its normal locus. The presence of some congenital deficiency in the resistance of the lacteal radicles may result in this phenomenon.

It is very easy to see how any growth, such as a neoplasm, an aneurysm, or constricting bands, might, by increasing the pressure within the thoracic duct at any portion of its course, result in the extravasation of this milky fluid into the abdomen. Most conditions acting by pressure on the thoracic duct carry a certain pathology and give definite symptoms indicative of their presence. Certain chylous cysts may have their origin in a similar manner from back pressure. Angiomatous disease of the lymph vessels might also cause the condition.

The *filaria sanguinis hominis* seen but rarely in this country has

been known to act as the causative agent. The symptoms may be so slight as to be overlooked, and the condition is only recognized in many cases by accident. In other instances the pathological process which causes the pressure on the ducts produces definite symptoms. The harsh, brassy cough of a thoracic aneurysm, the constant intrathoracic pain, the dyspnea, the tracheal tug, and the dilatation of the superficial veins of the chest will call attention to this condition. Physical examination of the chest will reveal an aneurysmal bruit, delay in pulsation in the vessels distal to the aneurysm, and a distinct expansile pulsation. The latter symptom, as I have personally verified, is sometimes absent.

The fluoroscope and skiagraphy, especially by the stereoscopic method in connection with the history and the physical examination will clear up the diagnosis of this causative pathology. Likewise, an intrathoracic tumor may be cleared up in the same way. Locally the presence of an ascites which is persistent and which on paracentesis or incision of the abdomen shows milky white fluid, makes the diagnosis. The course of the affection is afebrile and often does not materially affect the nutrition of the individual unless the causative factor is producing serious symptoms. By the absence of fever it may be differentiated from tuberculosis of the peritoneum prior to the discovery of the milky fluid. When the presence of this fluid is determined, the diagnosis becomes clear.

In case of filariasis the presence of other lesions, as elephantiasis of the limbs or scrotum, chyluria with ascites not otherwise explained, strongly point to this parasite as the causative factor of chylous ascites (peritonitis).

The filaria can usually be found in the blood, but it should be remembered that sometimes the filaria is only active in the blood at night, and, therefore, before a negative report is made, blood for examination should be taken at different hours of the day and night.

Boston⁴ tabulated 128 cases, some of which were probably pseudo-chylous and due to tuberculous or cancerous degeneration.

Edwards¹² collected 155 cases of adipose (chyliform or pseudo-chylous) and genuine chylous ascites.

Only 3 of Boston's cases were filarial. In only 11 was there demonstrated to be obstruction of the thoracic duct, and in only 7 was the duct of the receptaculum chyli ruptured. Otherwise, the point of leakage was not determined. Four of these were in combination with mesenteric cysts, all in young children. Chylous fluid has been found in the peritoneal cavity in several cases of the series of mesenteric cysts, in addition to these four, and in one case of multiple thin walled cysts, in

which the receptaculum and the first part of the thoracic duct were obliterated. The cysts were ruptured at the merest touch.⁴⁶

Thus it is likely that many cases of chylous ascites especially those of no tendency of reaccumulation and no demonstrable lymph trunk obstruction, originate as chylous cysts of the mesentery, and rupture into the peritoneum after the leak in the lacteal has closed.

The prognosis depends entirely upon the causative factor. Some of the simpler forms and those of congenital origin may continue without causing the patient any discomfort. Filariasis may be eradicated from the vessels in some instances and a recovery occur, but such a result does not always follow. The cases due to neoplasm and those dependent upon aneurysm are usually grave, and recovery will only take place when the causative factor can be removed.

Treatment.—Very little can be offered these patients in the way of therapeutics. Surgical removal of bands, either congenital or acquired, or of operable benign and malignant neoplasms offers something. For aneurysms of the type causing this affection, rest and remedial agents as proposed by Tufnell offer the most.

Formerly the only treatment afforded to persons infested with filariasis was entirely symptomatic and nothing was proposed to drive the parasite from the blood.

It is only within recent years that any effort has been made to attack these parasites in their almost inaccessible stronghold in the blood or lymph vessels.

Rogers³⁴ reports his experiments in the treatment of this affection by the intravenous injection of soluble salts of antimony at Puri in Orissa, at which point the disease is very common.

Ten cases of filariasis were treated by this method. From five to eleven injections were administered.

In only two to six of the injections was a full dose of 5 c.c. of a 2 per cent solution of sodium antimonium tartrate given. Two cases received 1 to 500 colloid antimony sulphid which was used in preference to tartar emetic because it is less toxic. The preliminary dose of either preparation in $2\frac{1}{2}$ to 3 c.c., increased $\frac{1}{2}$ c.c. at each injection until the maximum of 5 c.c. is reached. These injections are given daily for eight days, subsequently every alternate day.

Rogers claims that safe doses of the drug appear to produce a definite diminution of the number of filarial embryos in the peripheral blood. The effect on the adult worms in the lymph channels or on the symptoms resulting from their presence is as yet undecided. He thinks sufficient benefit has been obtained to investigate the subject further.

MESENTERIC CYSTS

The term mesenteric cyst is employed to describe certain cystic growths which develop within the folds of the mesentery.

Even those cysts which are almost entirely retroperitoneal are included in this group. The reason for this lies in the fact that cysts growing within the mesenteric folds may separate the layers and acquire attachment to the cellular tissue of the retroperitoneal space.

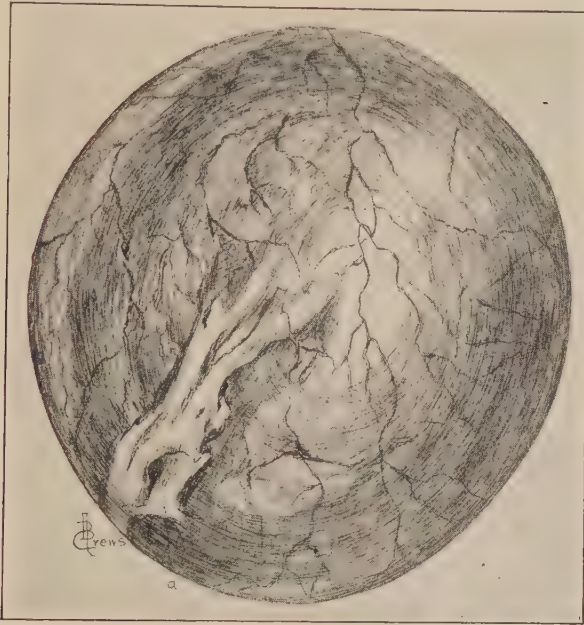


FIG. 60.—CHYLOUS CYST OF THE MESENTERY.
(Case of Dr. W. H. Axtell.)

On the other hand cystic tumors taking their origin from the retroperitoneal tissue may extend by pushing apart the mesenteric folds and in this way become completely enveloped by this structure. Cysts involving the omentum or developing within its folds are also included. Some of the cysts developing from the intestine and growing within the mesentery with propriety may be included in this group.

Mention of mesenteric cysts was made in the seventeenth and eighteenth centuries, but the communication of Portal in 1803 was the first effort to classify them.

In 1886 Augegnauer⁵ collected 18 cases out of 90 tumors of the mesentery. In 1897 Moynihan brought the total up to 113. Dowd²³

added 32 additional, a total of 145 cases. A number of these reports are incomplete.

Axtell⁷⁰ reports a case of chylous cyst of the mesentery which apparently followed an injury. He had been well previous to this injury and his abdominal symptoms appeared a few days later (Fig. 59).

VARIETIES OF MESENTERIC CYSTS.—These cysts are divided into four groups:

- A. Neoplastic
 - 1. Simple serous cysts
 - 2. Sanguineous cysts
 - 3. Chylous cysts
 - 4. Adenocysts
- B. Congenital
 - 1. Omphalomesenteric cysts
 - 2. Dermoid cysts
- C. Parasitic
 - Hydatid cysts
- D. Degenerative
 - Cystic degeneration of malignant growths

In Group A are classed all cysts of new formation not clearly congenital in origin. (Some of these may be of debatable origin.) These are serous cysts, sanguineous cysts, chylous cysts, and glandular cysts (adenocystoma).

Group B embraces those growths which are clearly of embryonal origin, the omphalomesenteric and the dermoid cysts.

Group C includes all parasitic cysts of the mesentery and of these hydatid cyst is the best example.

Cysts in Group D are not really cysts at all, but simply fluid accumulations in malignant tumors due to cystic degeneration.

In the above clinical classification it may be well to distinguish clearly between a cyst or an accumulation of fluid within a sac and a true cystoma. The term cyst as generally employed does not necessarily indicate that it is the result of any new formation of tissue. This is evident in degenerative cysts, retention cysts, and parasitic cysts. On the other hand the term cystoma or cystoblastoma of some writers clearly indicates the presence of new tissue formation.

True cystomata usually arise in structures which contain epithelium and if they develop elsewhere they are conceded to arise from cell rests or misplaced embryonic tissue. Some of such displacements develop true teratomata. Dermoids partake of this character.

Cystic formations as a rule develop under the following conditions and cysts of the mesentery are probably no exception. They either develop from simple cell inclusions of embryonal origin or grow from existing glandular structures and become isolated early in their developmental period. They may also form as retention cysts as when the duct of a secretory gland becomes occluded wholly or in part. Mucous glands in the intestinal wall may form such cysts. They may also form from hemorrhages into the cellular planes which may remain fluid, as in blood cysts, or partial absorption occur leaving a serous accumulation.

Cystic formations may result from the action of a parasite upon the tissues as in hydatid cysts.

Cystic accumulations also form as the result of cystic degeneration in malignant neoplasms. These are not true cysts at all but simply a phase of the malignant process.

Neoplastic.—*Serous Cysts.*—This is one of the simplest of all the cysts in its formation, but often one of the most difficult to explain satisfactorily as to its origin.

Some observers account for such cysts by stating that the serous fluid accumulates between the layers of the connective tissue, which becomes packed together so closely that absorption does not occur. That such accumulation of fluid may take place at points where no sac is normally present from prolonged pressure or irritation cannot be denied. In connection with the mesentery, however, such causative action cannot play a part. Others claim that such a cyst may follow considerable accumulations of blood which is absorbed with the exception of the serum and a small amount of detritus. One of Eve's⁷¹ cases seems to be of this type. The possibility of such occurrence must be admitted, but that it is a frequent source of the formation of cysts seems doubtful. It appears possible that between the original layers of the omentum where fusion has not completely taken place, serum may accumulate and the endothelioid cells of the peritoneum act as the secretory lining of the cyst.

Some observers strongly hold to the view that simple cysts of the mesentery are simply displaced hydatids of Morgagni, those small thin walled structures so frequently observed about the fallopian tubes. A strong argument in favor of this view is the monolocular character of serous cysts and the resemblance of their walls to the cysts of Morgagni. It must be admitted, however, that the cysts of Morgagni are exceedingly mobile, often with a long slender pedicle and that such a cyst might easily be transplanted some distance from its normal location.

Sanguineous Cysts.—Sanguineous cysts may form in the same way as serous cysts, or they may develop as true neoplasms from the endo-

thelium of the blood-vessel. Some writers contend simple serous cysts might easily occur without a secreting membrane either endothelial or epithelial.

Dowd²³ concludes that sanguineous cysts appear to be preformed cysts into which hemorrhage has taken place, and that hematomata of the mesentery should not be described as cysts. It is certain that hemorrhage does occur into preformed cysts, but these always have a wall of greater thickness than those recognized as blood cysts.

Chylous Cysts.—These cysts are more frequent than any other form of mesenteric cysts. Their exact origin is not clear. Some have claimed that they form as the result of angiomatous growths. Others claim that they develop from obstruction of the lymph channels, while still others, notably Dowd,²³ conclude that they are due to rupture of chylous vessels into a preëxisting cyst.

Of these three theories it would seem that lymphangiomata would be the most likely explanation of chylous cyst formation.

Adenocystoma.—This form of cyst contains true glandular structure of epithelial type, and either develops from preëxisting glandular structure or from sequestration of these elements from their normal site. Dowd's case is of this type and he goes to some length to prove from its characteristics and from its histologic formation that it was derived by sequestration from the ovary. His report shows the cyst to be a multilocular cyst adenoma of the transverse mesocolon, which in structure and contents resembled very closely the oöphoritic adenocystoma. The cases reported by MacDonald and by Morton appear to be of the same type.

Congenital.—*Omphalomesenteric Cysts.*—These cysts are evidently of embryonic origin. Their origin is more easy of explanation than any other. Such tumors develop from the remains of the omphalomesenteric duct which may become closed at each extremity leaving a central portion which may become distended and grow as a cyst.

The structure of this variety of cyst is identical with that of the intestine. The sac is composed of a serous covering, a well-formed muscular structure in two distinct layers, the internal being circular in direction, the external fibers running at right angles to the circular ones.

There is also a well-informed epithelial lining of secretory (mucous) cells separated from the muscular layer by a basement membrane of fibrous tissue with a typical muscularis mucosæ. These cysts are unilocular and contain mucin in considerable quantity.

From Studgaard's⁷² report it is evident that his case was of this type, both because of its funnel-like stem and from its histological picture.

He states that: "The cyst wall was, therefore, in perfect agreement as to structure with the wall of the intestine, save that in the latter we do not find an irregular hypertrophy of the tubular glands with degeneration of the superficial layer which was shown here."

Eve ⁷¹ described a cyst removed from the mesentery of the jejunum which showed in its wall three distinct muscular layers. The section showed the middle layer cut transversely and the other two longitudinally.

The cyst of the appendix which is seen very occasionally is of the same type.

The same is true of cysts of the urachus.

Dermoid Cysts (Teratomata).—These cysts are epiblastic growths due to embryonal cell inclusions and are fully explained by Cohnheim's hypothesis.

Most of the other epithelial lined cysts, including that of Dowd described under Adenocystoma are probably due to fetal remains, probably of aberrant ovarian tissue. Such cysts always contain mucin or pseudomucin, and are in many instances multilocular. Dermoids may occur in very young girls, when they are certainly of embryonal origin. In some adults, however, a few cases have presented containing in addition to the hair and nails usually seen, bones, and almost a whole skeleton in a few instances. There is a possibility in such case of the origin occurring from an ectopic gestation in the woman herself.

These dermoid cysts always have a very thick wall containing a grumous, mucilaginous, brownish material with certain fetal structures as hair, teeth, bones, etc. These cysts are usually simple, although sacculatation may be present.

Parasitic.—*Hydatid Cysts.*—The origin of cysts of this type has been conceded for many years to result from the presence of *Tænia echinococcus*. The hooklets from this form of tapeworm become attached to the tissues and each forms a brood capsule within a limiting membrane. From these brood capsules are formed daughter cysts. These small cysts make up the large mother cysts. The capsule of such a cyst consists of two layers. One of these, the external, is composed of elastic fibers and delicate connective tissue. The internal is made up largely of a homogenous structure carrying a water vascular system.

These cysts are always multilocular and if punctured they form other cysts wherever they are attached.

Nannotti ⁷³ collected 29 cases of this type, 16 proved anatomically and 13 others proved clinically. In several cases there were hydatids elsewhere.

The positive diagnosis depends upon the peculiar anatomical structure and the demonstration of the hooklets under the microscope.

Degenerative Cysts.—Degenerative cysts are sometimes found in uterine fibromyomata, in sarcomata of various types, particularly in the retroperitoneal tumors and may also develop in connection with secondary carcinomata involving the peritoneal tissues.

These cysts sometimes assume considerable proportions. They are pseudo cysts rather than true cystic growths in many instances.

The same treatment is applied to these growths that would be applied to the tumor in which they are found.

Symptoms of Mesenteric Cysts.—Slow growing mobile tumors appearing in the abdomen, particularly if originating in the upper portion, should excite the suspicion of mesenteric cyst.

The symptoms usually presented are digestive disturbances, malassimilation, malnutrition, discomfort from pressure. As the mass develops it may be made out as an elastic fluctuant growth, movable as a rule, although sometimes fixed. It may usually be distinguished from ovarian or pancreatic cysts by its location, from splenic enlargements by its rounded shape and absence of a notch, from renal tumors by the character of the urine and because it cannot be pushed into the loin. Ureteral catheterization with pyelography will usually tell positively. Pneumoperitoneum may be of great value. The laparoscope is of less value because of the ease with which a laparotomy may be done, and it will usually be indicated in any event.

Hydatids are differentiated by the peculiar tremor or thrill which is observed. The blood will show eosinophilia which is so characteristic of parasites.

Prognosis is serious though many recover after removal.

The treatment of these growths is essentially operative. Tapping is rarely employed at present though it was much used in the past. Hydatids should never be tapped since transplants are certain.

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CHAPTER X

TUMORS OF THE PERITONEUM

For the sake of clarity, tumors of the peritoneum are divided into primary and secondary.

In the first group will be considered the comparatively small number of neoplasms which are proved clinically and pathologically to originate in the peritoneal structure itself.

Secondary involvement of the peritoneum by growths developing in one of the adjacent abdominal or pelvic organs is of comparatively frequent occurrence.

Metastatic implantation from malignant tumors arising in distant organs also occurs frequently.

Neoplasms arising from the endothelium of the peritoneum or in its basement membrane are considered separately from those taking their origin in the retroperitoneal space. The latter are considered under the head of retroperitoneal tumors.

Those developing within the layers of the mesentery are classed as true peritoneal tumors. It may be contended that in effect the tissue between the layers of the mesentery is really retroperitoneal. However, the relationship of the tissues in this structure is so intimate that it becomes extremely difficult to differentiate clinically the point of origin of such growths; hence they will be considered as primary peritoneal tumors.

Primary tumors of the peritoneum, because of their infrequency and the difficulty with which they are recognized before the invasion of other structures has occurred, do not assume great clinical importance.

Primary retroperitoneal growths and secondary peritoneal neoplasms, on the other hand, are of considerable clinical interest.

Primary neoplasms of the peritoneum are classified clinically as benign and malignant.

The benign growths of this structure usually assume the mature types of connective tissue and are quite rare. Lipoma, fibroma, and myxoma have been observed. Some of the higher types of connective tissue tumors grow from this structure. Among these may be mentioned the angiomas developing from the blood or lymph vessels.

The lipomata arise either from the epiploic appendages or from

the omentum. They are extremely infrequent and as a rule only produce symptoms from their size. Peritoneal lipomata with small attachments sometimes become detached by tearing their pedicles, and under such circumstances are found as free bodies. Sometimes these become calcified.

The cases of fibromata and myxomata reported as arising from the peritoneum are of doubtful nature. They may develop from the basement membrane lying between the two folds of the mesentery. Undoubtedly the larger number of this type of growths spring from the retroperitoneal space and push forward between the mesenteric folds.

Vance¹⁴ gives report of 27 cases collected for the literature, as follows:

	Number of Cases	Deaths	Mortality Rate
Fibroma	9	1	11.1
Sarcoma	7	6	85.7
Lipoma	2	0	0.0
Myxofibroma	2	0	0.0
Carcinoma	1	1	100.0
Lymphangioma	1	0	0.0
Tuberculoma	1	1	100.0
Cholesteatoma	1	0	0.0
Hematoma	1	1	100.0
Myxoma	1	1	100.0
Large spindle-celled growths	1	0	0.0
Total.....	27	11	40.7

FIBROMA

This type of tumor is quite rare in this situation. Greer⁷ collected 33 fibromas of the mesentery from the literature to 1911. He credits Horstius (1578 to 1636) with the first report of mesenteric tumor found at necropsy. The first fibroma was reported in 1824 by Brichteau.

According to Bigelow and Forman³ 100 solid mesenteric tumors have been recorded.

Fibromata may occur as true fibromata, of which there are two types, hard and soft, or as mixed tumors, fibromyomata, osteofibromata, fibrochondromata, or fibrosarcomata.

In their origin these tumors, when pure, usually spring from the retroperitoneal connective tissue and are primary growth (Fig. 61). They are all of mesoblastic type. Because of the loose type of connective tissue formation in this locality they are of the soft type, hence grow to considerable size. In their gross appearance and because of their smooth outline and softness they may be mistaken for sarcomata.

A number of cases of fibromyomata are recorded in the literature. Such a growth must arise from some structure in which both non-striated muscle and connective tissue are normally present. Such growths may originate in the uterus as leiomyofibromata and become sequestered, to obtain attachment in the mesenteric folds, or they may arise from intestinal anlage and the latter is the most likely occurrence. These tumors are of slow growth, attain considerable size because of their internal structure and also because of the looseness of the surrounding tissue which permits them to advance. They are usually painless and only cause symptoms because of their size and from pressure upon neighboring structures as bladder and intestine. These tumors may be

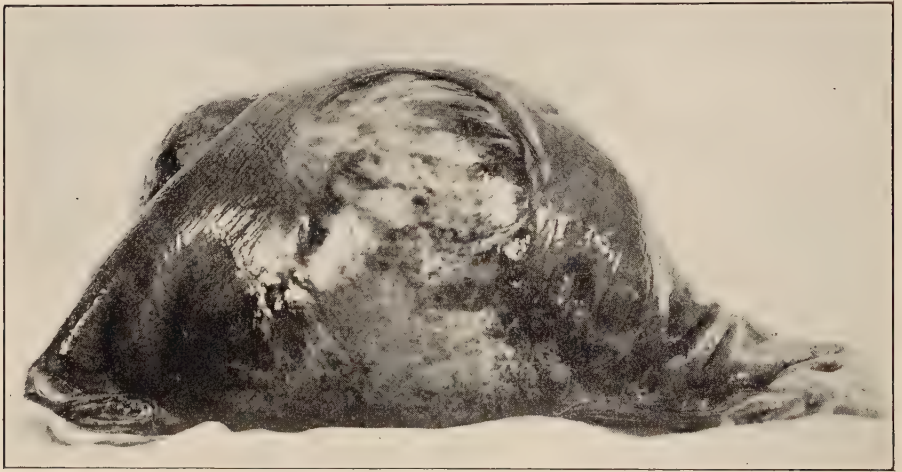


FIG. 61.—FIBROMA OF MESENTERY.
(Author's case.)

felt by the individual long before he consults a physician.

An injury sometimes calls attention to such a growth which has existed previously unnoticed. Frequently a diagnosis is not made as to the location of the growth until operation is performed. The recognition of the mass should be easy since it usually reaches some size. The growth is usually single. That is, there are no nodules unless it be a sequestered uterine growth. It is usually encapsulated, lies behind the intestines or pushes them aside as it grows forward. It does not move with respiration although it may have a slight mobility. Its base seems to be attached. It is firm, elastic, but does not fluctuate. Thus it can be distinguished from a cyst.

By the use of pneumoperitoneum the origin of the growth may be correctly interpreted.

Renal tumors also spring from the posterior peritoneal space. They may be recognized by the hematuria, by their close relation to either loin, and by pyelography.

Pancreatic tumors are more difficult to distinguish from mesenteric fibromata because of their situation. They are located at a fixed point, the pancreatic region. Often loss of pancreatic function can be made out. The intestine lies over or around a pancreatic growth, or the stomach covers it. The stomach and intestine move away from it on manipulation or during physical or x-ray study, while the gut may be closely attached to the fibrous neoplasm.

Hepatic enlargements are identified by their continuity with the liver, increased dulness, moving with respiration and with the liver.

Intestinal tumors are more mobile, more tympanitic, cause more interference with intestinal function. A roentgenogram is very valuable.

Pelvic tumors, as ovarian cystomata and fibromata and also uterine tumors spring from the pelvis and their relation to the uterus and adnexæ can be made out. Here again pneumoperitoneum is a valuable diagnostic measure.

Such tumors are histologically benign and may continue for years without causing much distress. They are potentially malignant clinically because from their size alone they may interfere with the functions of other organs sufficiently to endanger life. Vance¹⁴ gives 40.7 per cent mortality in 27 cases. In 13 of these in which resection of the intestine was necessary, the mortality was 46.15 per cent. The high mortality in this group of cases is due to delay in reaching treatment.

The treatment is surgical since no other known method of cure can be offered at present. Radium may offer something, but with our present knowledge early removal is the best method of dealing with such neoplasms.

The dangers from resection are hemorrhage, shock, embolism, phlebitis, gangrene of the intestine, a leak at the line of suture, peritonitis.

Shepherd¹² reports resection of one such neoplasm with eight feet of intestine, and recovery.

RETROPERITONEAL LIPOMA

These tumors may appear as true lipomata with just sufficient connective tissue stroma to make a stable structure. They may also appear with a considerable amount of fibrous tissue and the term fibrolipoma is justifiable.

The large fatty masses of omentum sometimes seen do not constitute true lipomata.

The growths usually appear in adult life. They belong to mature types of connective tissue growths. Rarely they show embryonal elements, when they must be classed with the sarcomata and are malignant neoplasms. They take their origin at the root of the mesentery and while primarily probably retroperitoneal, they advance between the layers of the mesentery and are really true mesenteric tumors. They sometimes occur with similar growths in other parts of the body (multiple lipomata).

They usually grow slowly and are of moderate size, but may grow to considerable proportions, pushing aside the viscera and causing disturbance from their bulk. They may infiltrate almost entirely around the intestine in some rare cases and partially separate the serosa from the other layers of the intestine.

They are painless except when their size causes pressure upon a nerve or when the nerve is pulled upon as it passes round the growth by the muscular movements of the body. They are semifluctuant in feel, nonmobile as a rule, easily mistaken for a mesenteric cyst.

The diagnosis may be made of retroperitoneal or peritoneal tumor by its relation to the intestines, which always lie in front of the swelling. If the growth reaches large size and its surface approaches the anterior wall the intestinal coils lie around the center, and there is an area of dulness surrounded by tympanitic resonance.

The prognosis is uncertain depending upon the pressure effects of the growth and its visceral attachments. It is less serious than some of the other tumors. It is pathologically benign and clinically only becomes malignant from its size, location and pressure effects. When extensive surgery is necessary for its removal, it becomes a dangerous condition.

The treatment is conducted along general surgical lines. Enucleation is indicated when the damage to the intestines incident to its removal is within the bounds of safety. After the removal, hemostasis should be complete. The peritoneum which has been opened to release the growth should be sutured and as little dead space left as possible.

It is scarcely possible to separate peritoneal lipomata from those of the intestine.¹³

Vaccari¹³ finds only 69 cases of lipoma of the intestine reported to date. His own case was a man of sixty years of age, who came to operation with the diagnosis of intestinal occlusion. This proved to be an invagination of the last portion of the ileum. The intestine resisted attempts to relieve the invagination, and required resection with terminal closure of the stump and a side-to-side ileocolic anastomosis. Death oc-

curred in two days. The autopsy revealed diffuse, fibrinous peritonitis. In this case all the chief symptoms of invagination were absent. He did not see his patient until about sixty hours after the onset of the invagination. He points out that the muscular tissues of the intestine constitute a barrier to the development of lipomata arising in the intestinal walls. Lipomata which originate in the submucosa grow toward the intestinal lumen, and those which form in the subserosa have a tendency to rise from the side of the peritoneal cavity. In examining the resected portion of the intestine in the case reported, he discovered that there were two distinct lipomata of the intestinal wall, one subserous and the other submucosal, which were separated by a double stratum of more or less altered smooth muscle fiber, circular and longitudinal.

Although several writers have suggested that lipomatous new formations and true lipomata may be formed through metaplasia or degeneration of connective tissue cells, Vaccari did not observe any cellular elements demonstrating such a transition.

HEMANGIOMATA

This type of tumor is very rare. These growths spring from the blood-vessels and usually appear as simple or cavernous nevi and are congenital in origin. Occasionally they develop later in life. They are sometimes solitary and sometimes multiple. They may spring from the mesentery, from the intestine, stomach, and the liver.

The author reported to the Southern Surgical Association, Atlanta, a neoplasm of this type involving the stomach. It was treated by partial resection of the stomach with recovery. The patient is still living, in good health, without a recurrence.

Helvestine reported 14 cases of hemangioma of the intestine. Most of the growths involve the small intestine. They develop in the vessels between the circular or longitudinal layers, or under the serosa.

Hemangiomata appear to be causative of intestinal obstruction in three cases of his series, 21.4 per cent, one by the size of the tumor and one because of intussusception. The third case gave symptoms of chronic obstruction.

The collected cases showed hemorrhage in 28.6 per cent, 4 of 14 cases. It was the cause of fatality in 2 instances.

These tumors are benign in their inception, but may undergo malignant change. They are usually small in size.

The clinical evidence of the presence of such tumors is small and

depends upon the location of the growth. My own case gave evidence of gastric disturbance, discomfort after meals, and hematemesis. The latter was not frequent. Some cases involving the intestine give rise to blood in the dejecta and occasionally result in intestinal occlusion.

The diagnosis of the exact nature of the growth may scarcely be determined prior to operation for its relief. The presence of a tumor may be ascertained prior to operation although its exact nature may only be determined later.

The diagnostic signs are similar to other mesenteric growths except from the smaller size of these growths the symptoms are more obscure. This type of growth is essentially benign. The course is slow and the discomfort is usually slight. Hemangiomas of the intestine may result in invagination. Treatment consists in removal of the neoplasm upon its recognition when this is possible. Cauterization has also been employed.

LYMPHANGIOMA

Angiomas involving the lymphatics of the peritoneum are very rare, and chylous cysts sometimes develop in connection with this class of growths. They may develop to considerable size, in some instances holding several liters of fluid. These cysts are thin-walled and have a limited blood supply, as a rule. The fluid contents consist of a yellowish white milky fluid, which resembles chyle in appearance and general characteristics. The presence of fat droplets in suspension, fatty acids, cholesterolin and other constituents of chyle, permits the differentiation of this type of cyst from serous cysts, and also chylangiomas from lymphangiomas proper.

In their development these cysts may push the intestine in front of them, and occasionally the intestine may become adherent to the cyst. The cyst may in other cases push the intestines aside and present against the abdominal wall. This is particularly likely to occur when the growth is of large size.

Whenever the intestine overlies the cyst a tympanic note is elicited on percussion. When, however, the intestine is pushed aside the percussion note is dull. Very occasionally in the large cysts of this type fluctuation may be elicited, but there is never the tremulous thrill of the echinococcus cyst.

Serous cysts are sometimes seen in the mesentery, but more frequently in the peritoneum of the broad ligament, near the ovary and tube where they probably spring from fragments of the wolffian body.

Occasionally a cyst develops from the remains of the omphalomesen-

teric duct. Cysts of this type are lined with epithelium and grow to considerable size.

The symptoms presented by a chylous cyst are for the most part due to the effects of pressure. Because of the location of these cysts and their slow development, the symptoms come on very slowly and pain may be slight, very late in making its appearance, or may be entirely absent. If situated where pressure is produced upon the vessels, and when sufficiently large, swelling and edema may occur in the limb of that side. Occasionally pressure may produce an intestinal obstruction, and this may be the first evidence of the presence of a growth.

The first evidence given by most of these cysts of their presence is palpation by the patient of a mobile or a fixed mass within the abdomen. The presence of a fluctuating retroperitoneal growth yielding chylous fluid on aspiration is a conclusive diagnostic sign.

The retroperitoneal position of the growth may not be determined before section is performed, but the skilled diagnostician may by physical examination be able to make out by percussion and palpation whether the growth lies beneath the peritoneum. If the intestine lies over the growth superficial percussion gives a tympanitic note, while deep percussion elicits dullness. Under these conditions the cyst may be but slightly mobile. If, however, the growth is large enough to push the intestine aside and lie against the abdominal wall, dullness will be elicited over it, and tympany all around it. The cyst may in such a case develop considerable mobility and fluctuation may be detected. This class of growth will show no connection with the uterus or adnexa, which will aid in differentiation from ovarian cyst and uterine neoplasms. In cases of doubt, the employment of radiography with pneumoperitoneum will be of great service in locating the site of the mass.

The treatment is surgical and the procedure will be determined by the size and relations of the growth. Excision is the operation to be employed when it may be safely accomplished. Marsupialization will rarely be advised.

ENDOTHELIOMA

The most important of the primary growths springing from the peritoneum is the so-called endothelioma. This growth probably arises from the endothelium of the peritoneal surface and some authors believe that it takes its origin from the endothelium of the lymphatics. The presence of mucous cells which have been found in some cases has been considered as evidence of its epithelial origin. It has been called primary carcinoma of the peritoneum by some.

Warthin¹ claims that only those growths which arise from the columnar epithelial cells of embryonal inclusions of intestinal anlage should be called primary carcinoma of the peritoneum. This latter is a very rare condition.

Endotheliomata appear as flat plaques or nodules more or less confluent. The plaques may be joined by cordlike bands. Occasionally there is more elevation of the nodules. A number of minute flattened structures are sometimes grouped about a larger mass. They are white in color and soft in consistency. Rarely they may be firm. The adjacent peritoneum is somewhat thickened. Occasionally one of the larger masses is softened in the center very similar to the caseation of a tuberculous nodule. Sooner or later there is always present a serous exudate. This may contain some fibrin and occasionally it is blood stained.

The microscopic picture is that of connective tissue stroma enclosing strands or cordlike groups of cells. These are often low columnar cells arranged upon a basement membrane. This gives to the tumor the appearance of a tubular gland. Some of the larger plaques are devoid of surface epithelium while others smaller in size show many layers of cells.

There is little tendency to metastasis into the solid organs, but secondary deposits are found in the other serous membranes. The growth is identical with the flat tubular endothelioma of other serous membranes. This growth is progressive and probably malignant. The tendency is to spread on the peritoneal surface rather than by infiltration into the tissues.

The symptoms presented are not distinctive. Loss of flesh, fullness in the abdomen, some evidence of ascites without febrile reaction point to the possibility of this neoplasm being present.

The diagnosis will not be made before abdominal section in many instances. Rarely there may be present palpable masses within the abdomen, but this is not the rule.

The treatment of this affection is not satisfactory. Operative treatment may be beneficial in the very early stages, particularly when discovered when operating for some other lesion. When the symptoms are sufficient to enable the recognition of this disease, dependence must be placed in the use of radium or deep x-ray therapeutics.

CARCINOMA OF THE PERITONEUM

The endothelioma is the only primary peritoneal growth which is similar to carcinoma. To all intents and purposes it belongs to this class of malignant neoplasms.

The very large number of secondary growths, however, are carcinomata. Among these growths are found certain colloid carcinomata and adenocarcinomata. Cancers which are primary in the intestine are almost all of this variety.

Colloid Carcinoma.—Colloid carcinoma is a variety of cancer seen occasionally, involving the peritoneum, the gastro-intestinal tract, the gall-bladder, the urinary bladder, the kidney, ovary, cervix uteri, salivary glands, and the bronchus. The terms mucoid carcinoma, gelatinous carcinoma, and malignant myxoma have been applied to it. The original term, given by Virchow in his description of this condition, colloid carcinoma, has met some objection because of the fact that the term colloid is now used to mean the gelatinous substance, containing iodine, which is found only in the thyroid gland, and the term mucoid is perhaps more descriptive of this particular lesion, since the structure is composed largely of mucin.

Development.—These growths may form in epithelial tissue, normally producing, or capable of producing mucus. These growths show delicate connective tissue stroma with large accumulations of mucoid material between the connective tissue. Most authorities regard the production of mucin in carcinoma as a degenerative change.

Ziegler claims that in intestinal cancer, the formation takes place in goblet cells normal to these structures. In some instances the colloid formation is present in the early stages in the development of the tumor, which would seem to point to its being a synchronous development in certain epithelial growths, and not necessarily a degeneration. This contention seems to be borne out in the fact that in metastatic growths of this type the secondary mass always contains the same gelatinous material. The origin of the colloid material is probably from activity of the epithelial cells, since the small droplets of mucin are observed within the protoplasm of the epithelial cells, and in the adenocarcinoma they are found within the acini (Parham).⁹ In the development of these growths the cell division is very slow, and these carcinomas grow very slowly notwithstanding the large amount of accumulation of colloid. The growth is very persistent, however, and these cases eventually involve the entire abdominal cavity and greatly increase the size and contour of the abdomen. The quantity of the colloid material varies very greatly. In small areas in such structures, masses of malignant cells with but little sign of colloid may be present. In other tumors colloid may be entirely found within the cells. There is usually present in these growths a tendency to the formation of acini. In their clinical history these growths show but little tendency to encapsulation. They are slow

in growth, and metastasis occurs less promptly. They expand by direct progression and infiltration. Contact transplants are frequently seen. The structure of the growth is extremely friable, and its vascularity is small compared to some other types of tumor. Growths developing in the intestinal tract have a tendency to expand through the walls and when this takes place there is prompt transplantation upon the peritoneum, which seems to favor the development of the growth. These tumors present the appearance of a light yellow jelly enclosed in a very delicate connective tissue structure. In some cases, especially those arising from the intestine, a large or small number of segregated masses are present, composed in large part of tapioca-like bodies. Like other slowly growing neoplasms of malignant type, these tumors are essentially fatal, and while progressing more slowly, nevertheless persist to a final destruction of the individual.

"Colloid carcinoma of the peritoneum usually presents a characteristic picture. The parietal and visceral peritoneum is studded with large and small soft glistening nodules. The cavity contains more or less jelly-like material, often round, hard, gelatinous bodies resembling tapioca or fish eggs. Adhesions may be present in abundance, matting together the intestine. The microscopic picture is one of an abundance of colloid with scarcity of cellular elements. These are usually of the columnar type and arranged in chains. Their protoplasm shows accumulating mucous droplets" (Parham).⁹

This structure is invaded secondarily in almost all cases of gastrointestinal growths of this type. In fact the larger number of cases showing peritoneal involvement are secondary, and the determination of the primary origin in some cases is difficult.

That primary involvement of the peritoneum does occur rarely, one must admit, but the type of cells within the peritoneal structure is not one that produces mucus. There is a slow but progressive involvement of the peritoneal tissues until every portion is involved in some cases, these gelatinous masses fastening themselves upon the liver, even invading the under surface and perhaps into the lesser peritoneal sac. We have observed in recurrent growths of this kind developing primarily in the colon, an infiltration of the abdominal wall in which ulceration extended from the gut through the parietes with the formation of fecal fistula. In this case the gelatinous masses involved all the structures of the wall.

Perhaps the larger number of cases of general peritoneal involvement develop from proliferating papillomatous cysts of the ovary. In the experience of the writer some of the cases in this particular group

progress more rapidly to a fatal issue than is usual in colloid carcinoma. Smith, quoted by Parham,⁹ in 1893, mentions a case lasting for nine years in which paracentesis was performed two hundred ninety-nine times. At necropsy a papillomatous tumor of each ovary was present.



FIG. 62.—COLLOID CARCINOMA WITH FECAL FISTULA.
(Author's case.)

Trotter, in 1910, reported a case originating in the appendix. There are a few cases on record in which spontaneous cures have occurred (Trotter, Lejars, and Eden).

Lejars claims that colloid carcinoma of the ovary is more malignant than that of the appendix.

Parham says, "It would seem possible to tell whether these cases

were of ovarian or extra-ovarian origin by chemical means. The gelatinous material produced by the cells of ovarian origin contains pseudomucin, soluble in alkaline solution, not precipitated by acetic acid, and stained by acid stains. There were thirty-seven cases of colloid carcinoma of the peritoneum of uncertain origin in this series. This is sixteen per cent of a total of two hundred thirty-two cases of abdominal carcinoma. Of fourteen patients traced, two were living and responding remarkably well to large doses of Roentgen-ray and radium. One patient had lived eleven years since operation and twelve were dead, seven less than one year after operation, two less than two years, one less than three years, and one less than four years."

Clinical characteristics are not many. The onset is slow, and the inception may follow an operation for the removal of an ovarian cyst, papillomatous proliferating in type. Or there may be some intestinal or gastric symptoms prior to the patient's attention being called to the change in the abdomen. The first thing noticed may be gradual, painless enlargement of the abdomen, with some loss of flesh in other portions of the body. There is a certain amount of anemia present and the patient complains of loss of strength. As time goes on prostration and cachexia are noticeable.

The course is afebrile throughout. The patient may discover palpable masses within the abdomen. Ascites is a frequent concomitant.

In cases involving the intestine, obstruction is not infrequent, and in some cases this first brings the patient under observation. In many of these patients the condition is recognized by accident.

Diagnosis.—This is made by the previous history, particularly of ovarian cystoma, also by the presence of a slow growing mass in the abdomen, with evidence of ascites. The fluid may be present in some cases in only small amounts. The absence of fever is a strong aid in diagnosis from tuberculous ascites. However, this elevation of temperature is sometimes absent in tuberculosis. Usually it appears as a constant but slight afternoon rise. Ascites from other sources can be differentiated by the symptoms of the causative condition, and by the presence of the irregular masses in colloid carcinoma. Cancer usually, but not necessarily, occurs in older patients than does tuberculosis.

Cirrhosis of the liver may occur at any age. Usually it appears in adults from thirty to forty years. It is slow in development and follows a history of alcoholic excess. Morning nausea and vomiting are of frequent occurrence. The vomitus may consist of mucus or blood. The patient may be robust with a distended abdomen which gives the usual signs of the presence of fluid. The liver dulness is perceptibly dimin-

ished. The veins of the abdomen are greatly enlarged if the symptoms have been present for some time. Hemorrhoids are nearly always present.

The onset of colloid cancer is also insidious. It usually develops in persons over forty years of age. Painless swelling of the abdomen with slow but persistent loss of flesh and strength without fever, points strongly to malignancy. A cancerous cachexia develops after a few months.

The abdomen is doughy in feel. The mass lies close to the parietes and is semisolid rather than freely fluctuant. Dulness is usual and masses may be palpable. There is no morning nausea, no vomiting, no hematemesis. Diarrhea and constipation may alternate. The stools may be bloody at times. The use of the more exact methods of examination may be necessary for correct differentiation.

Renal growths have a characteristic location and outline. They usually show a certain mobility.

Solid tumors may be distinguished by bimanual manipulation. They always show at some period of their development hematuria. Hydronephrosis may have been preceded by evidence of stone, as crystals, blood, and perhaps pus in the urine. Hydronephrosis develops slowly from above, may show recessions in size. It gives rise to a fluctuant tumor moving up and down with respiration.

Roentgenograms and pyelography will determine the origin of all renal masses. In hydronephrosis the stone may be shown.

Large ovarian cysts may be difficult to distinguish from a colloid carcinoma in a few instances (Fig. 63). They develop from below and reach considerable proportions. Often they are nodulated or irregular in outline. Ascites is frequently present.

A proliferating ovarian cyst may be the source of origin of a colloid carcinoma.

Pelvic examination may determine that the mass grows from the ovary. Anesthesia is valuable in making the examination. The fluid may be removed from the abdomen as an aid in diagnosis. The laparoscope may also be employed if other measures fail to differentiate the condition.

A roentgenologic study of the gastro-intestinal tract will often be of material service in diagnosis. This is particularly true if pneumoperitoneum is employed in the investigation.

Prognosis.—The prognosis is always grave notwithstanding the fact that a few cases are recorded where recovery occurred spontaneously or under treatment by radium or Roentgen-ray.

Treatment.—When this condition can be recognized sufficiently early to permit radical surgical removal, there can be no question that this offers the best chance for a cure. Following this deep x-ray and radium are to be employed with sufficient vigor to obtain the maximum

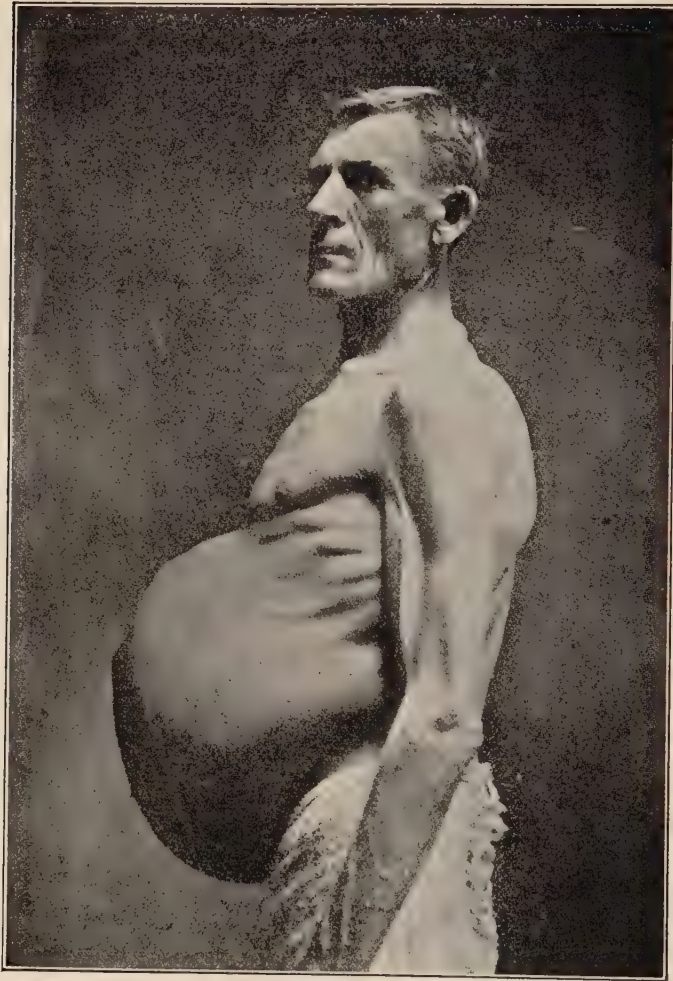


FIG. 63.—COLLOID CARCINOMA.
(Author's case.)

benefit. This treatment should be conducted by expert roentgenologists and radiologists and kept within the bounds of safety.

In cases where recurrence has taken place and in those in which radical surgery is impossible, radiation should be employed. In some cases the results have been most satisfactory.

Intercurrent complications such as intestinal obstruction should receive appropriate treatment.

Paracentesis, so long in vogue in the management of these cases, is only indicated as a palliative measure to relieve the pressure effects of ascitic accumulations.

ADENOCARCINOMA

These growths are secondary to primary tumors in the intestine as a rule. Sometimes they are secondary to adenocarcinoma of the mammary gland. They may also be one of the late results of this form of tumor of the fundus uteri.

Adenocarcinomata of the ovary may be the primary disease followed by secondary implantations into the peritoneum. Many such cases develop very rapidly following contamination of the peritoneal sac from the escape of fluid from a proliferating papillomatous cyst of the ovary. These cases run a very rapid course. Usually such secondary involvement of the peritoneum partakes somewhat of the nature of an inflammatory process. According to some pathologists it gives the clinical picture of a peritonitis and receives the name peritonitis carcinomatosa. There is no malady which is more fatal than such a secondary carcinomatous involvement.

The peritoneum becomes rapidly studded with carcinomatous plaques and nodules, varying greatly in size. The entire peritoneal structure may be involved. In some instances a considerable amount of thick albuminous ascitic fluid is present. In other cases the intestines are agglutinated together as in tuberculosis. These patients lose flesh and strength rapidly. They become markedly anemic and as the patient becomes smaller the abdomen becomes larger. This distention of the abdomen with ascites and carcinomatous masses is characteristic. Such masses are readily palpable at times.

There is a remarkable freedom from pain in some of these cases. When, however, interference with the fecal flow is present, pain becomes a serious complaint.

The author has observed such a peritoneal carcinomatosis following six years after a primary carcinoma of the left breast. Two years subsequent to the removal of the left breast a carcinoma of the right breast developed, no tumor being observed before, and was removed. Both of these operations were of the most radical type and no local recidivism occurred. Four years after the second operation peritoneal involvement took place and the patient lived more than a year afterward.

She suffered no pain whatever during this time. Her only complaint during this time was from the pressure effects of the ascites. Paracentesis produced temporary relief from this symptom.

The symptoms of such a case must awaken in the observer the belief that only an infectious process resulting from a living organism must be the causative factor. No other pathological process, except an infection, can account for all the phenomena presented by cases of this type.

The question arises as to what effect inflammation of the peritoneal structure has in stimulating an embryonal rest or embryologic tissue into activity. Undoubtedly any form of irritation might act upon an embryonal collection of cells to stimulate their growth. This might be active in the production of a neoplasm. It certainly has resulted in the production of certain cystlike accumulations about the peritoneum. Such occurrences are so infrequent, however, that this action must be considered as negligible. Any sort of prolonged irritation may by weakening the resistance of a part, render easy the location of a particular organism in the production of a certain disease.

If, therefore, one admits that cancer may be due to a living organism with selective tendencies then he must admit the possibility of malignancy becoming engrafted upon an inflamed or damaged peritoneum. It must be remembered that the less active the organism and the slower its development the more persistent are its effects and the more fatal the final result. Tuberculosis is of this type. Why may not cancer be the same?

A few words concerning cancer of the intestine may not appear amiss.

In 1904 (*Medical News*, May 21), the author presented the report of two cases of carcinoma of the intestine, and made an extensive study of the literature up to that time. In that study it was found that Hemmeter had collected 69,083 autopsies including those of a number of others, with 5,796 carcinomata, 8.4 per cent of the whole. Of these cases, 1296, or 22 per cent had suffered from carcinomata of the intestine.

DeBovis claims that one cancer of the large intestine occurs in every 300 deaths. He collected 426 cases of cancer of the large bowel in 2,500 cases of illness. He gives the proportion of carcinoma of the small gut as 6.3 per cent, of the sigmoid 11.9 per cent, colon 20.4 per cent, rectum 49.2 per cent, cecum and appendix 12.2 per cent.

The variety most often met is the adenocarcinoma. Spheroidal celled carcinoma, either of the scirrhus or medullary type is also found. The

adenocarcinomata rarely cause circular occlusion, but this is the usual termination of scirrhus (Fig. 64).

Any form of malignant disease by matting the intestines may interfere with fecal flow.

Carcinomata of the intestine rarely involve the mesenteric glands, which is contrary to expectation. Sarcomata, on the other hand, show secondary involvement of the mesenteric glands in 68.4 per cent of the cases, according to Jopson and White.⁴¹

As many as 10 per cent of the cases of cecal cancer collected were diagnosed as appendicitis. Many of the cancers were discovered at

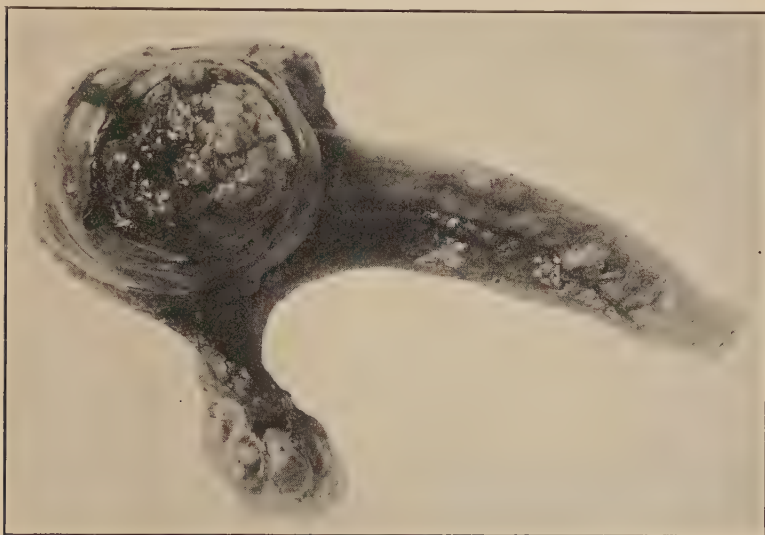


FIG. 64.—ADENOCARCINOMA OF INTESTINE.
Living 8 years after resection. (Author's case.)

operation for intestinal obstruction. The possibility of a malignant growth as the cause of intestinal obstruction must be constantly borne in mind.

In one of the author's own cases the carcinoma of the caput coli showed a rupture through the posterior wall from ulceration. An abscess had developed giving rise to elevation of temperature, pain, tenderness, and rigidity, with nausea and vomiting. Leukocytosis was increased. The attention of the patient had only recently been called to this condition. These symptoms were identical with those seen in acute appendicitis. Such symptoms indicate clearly the reason why so many of these cases are diagnosed as appendicitis.

SARCOMA

Primary Lymphosarcoma.—Primary lymphosarcoma of the intestine almost invariably involves the peritoneum secondarily.

It has been looked upon as a very rare neoplasm. In sixteen years, 1859-1875, not a single case is recorded in the Berlin Pathological Institute. ⁴⁶

Nine cases of lymphosarcoma are found in 21,358 autopsies in Wiener Krankenhaus from 1882 to 1893, inclusively.

Nothnagel ⁶² reports 12 cases of sarcoma. Smoler ⁷⁶ records 13, Treves 18, Jopson and White ⁴¹ 22 cases. Libman ⁴⁷ records 59 cases, Harte ³⁷ one in a child of five years of age, Robertson ⁶⁸ a case occurring in a child four years old, situated near the ileocecal valve.

Crowther ¹⁶ collected the largest number of recorded cases amounting to 122 in 1913. Huettl ⁴⁰ reports 9 cases. Graves, ³² presented 3 cases and collected 246 others, making a total of 249. The ages range from four to seventy-five years. Graves correctly contends that the term sarcoma should be replaced by the term lymphoblastoma as being more correct pathologically. Because of the universal use of the older nomenclature these terms are employed in this monograph, since it does not seem within our province to adopt a new nomenclature.

These growths usually begin in the lymph follicles, which apparently accounts for the early involvement of the lymphatic glands, a rare occurrence in intestinal carcinoma.

The serous covering of the intestine is involved and frequently is studded with nodules very similar to those of tuberculosis.

The presence of girdle ulcers with sharp steep walls is mentioned by V. Aschoff. ⁹¹

Rarely is the serosa perforated, retaining its protective resistance to which attention has been called previously.

The infiltration into the coats of the intestine interferes with its nervous mechanism and extreme dilatation may occur. Obstruction does not seem to be a prominent factor.

Prognosis.—This has been looked upon as a fatal affection. In the light of the more recent records it would appear that a considerable percentage live for long periods without recurrence after operation.

Treatment.—This consists in radical removal when it may be safely accomplished. Arsenic has been employed in the treatment. A number of cases respond to the use of radium and x-ray therapy. The combination of operation with radiation seems to give the best outlook.

Ewald ⁹⁰ mentions a case reported by Bessel-Hagen of a seven-year-

old boy, in whom during the course of a sarcomatous infiltration of the jejunum, there had formed an aneurysm-like dilatation of the size of a large man's fist. Jopson and White ⁴¹ mention a similar case.

Retroperitoneal Sarcoma.—This form of neoplasm is in the greater number of instances secondary, the primary tumor developing from the intestine, the ovary or the extremities.



FIG. 65.—RETROPERITONEAL LYMPHOSARCOMA.
(Author's case.)

In some instances following sarcoma of the testis, there is a rapid involvement of the retroperitoneal glands, and also frequently an extension into those of the mediastinum. The writer has met with instances of this type in which, following a sarcoma of the right testis, secondary involvement occurred in the retroperitoneal, lymphatic glands on the right side of the abdomen, while the glands on the left side of the chest were enlarged. There was no involvement of the glands upon the right side of the chest. While secondary growths in this region are of somewhat frequent occurrence, those of primary origin are rare.

In 1900, Steele,¹¹ reviewing the literature up to that time, recorded 96 cases of primary retroperitoneal sarcoma, including 5 of his own. Trout and Meekins,¹³ in 1920, cite 12 additional cases, including 2 of their own. In 1923, Andrews¹ reported in addition 28 proved cases from the Mayo Clinic, making a total of 142 cases. A case of my own, previously reported, but not included in either of the above reports, brings the total up to 143. This case was a lymphosarcoma (Fig. 65).

The type of growth most frequently observed is the small round-celled lymphosarcoma, which occurred in 10 of 28 cases recorded by Andrews. According to the same author, the next in frequency were the spindle-celled and the fibrosarcoma, each occurring in 4 instances. The mixed celled fibromyxoma, and myxosarcoma were each found in 2 cases. Small and medium round cell sarcoma, myxo-osteochondrosarcoma and giant-celled sarcoma were found in one, each; the latter was made up, for the most part, of tumor giant cells.

Clinically these growths vary considerably in density, size, and rapidity of growth. A number of factors enter into the determination of the clinical course of such neoplasms.

The more embryonic the type of cell prevailing, the more rapid the mitosis, the faster and more malignant its growth. It is generally accepted as a fact that the small round-celled sarcoma is the most rapidly growing and the most malignant of this class of tumors. Metastasis is far more frequent in tumors showing this cellular formation. This can be accounted for by the close relationship of the cells to the blood-vessels; practically, the cells of the tumor lie in juxtaposition with the vascular canals. Hemorrhage not infrequently occurs into these tumors, first, because of their rapid growth, second, because of their great vascularity. These round-celled growths spring either from the soft connective tissue in the retroperitoneal space, or from the lymph-nodes. While encapsulated at first, the mitosis is often so rapid that they soon in many instances infiltrate through the capsule and invade neighboring structures. The rapid growth and the vascularity usually impart to these growths a semisolid or even fluctuant feel. They also show such active tissue change at times that they feel hot to the examining hand. Sometimes, on account of the fluctuant feel, the heat and, when approaching the surface, the redness, they are mistaken for inflammatory conditions.

Of the round-celled growths, those which are pigmented progress the most rapidly to a fatal issue.

The cellular arrangement in these round-celled growths is interesting. The round cells are very numerous, massed closely together, show single

deep staining nuclei with little cytoplasm. The capillaries are very abundant, often composed of a single layer of endothelial cells. In some instances the blood seems to lie in direct relation to the tumor cells. Mitoses are frequent. The stroma is often difficult of demonstration.

It is well known that the more a tumor cell is differentiated, and the fewer mitoses, the more mature the type of tissue entering into its formation, the less malignant it is.

As might be expected those growths showing differentiated spindle cells and also the fibrosarcomata made up either of large round or spindle cells with a considerable amount of connective stroma, usually grow slowly. They show comparatively few mitoses, are surrounded by a firm capsule, only occasionally infiltrate beyond it, and are firm to the feel. When removed they do not recur so promptly as the class first described. Metastasis sometimes occurs but nothing like as frequently as does the small round celled growth.

Both the large and the small spindle cells are arranged in bundles around the capillaries with definite well-developed interstitial connective tissue. Cut longitudinally, these cells shows the typical elongated, somewhat pointed cells, oval or spindle-shaped nuclei. The blood-vessels are more fully formed than in the round-celled growths, which are much more malignant than are the spindle-celled forms.

Those forms of sarcoma which present the mature types of connective fibrous tissue, cartilage, bony or mucous tissue are slow growing and less malignant than the previously mentioned types. They are named according to the type of tissue preponderating, fibrosarcoma, chondrosarcoma, osteosarcoma, myxosarcoma. Examined in certain portions only, these growths may be mistaken for nonmalignant neoplasms. The examination of sections taken at several points along the advancing margin will show rapidly growing malignant cells. The least malignant of all these sarcomata is the giant celled growth. They are infrequent in this location, since most of such growths develop in connection with bony structure, although this has its exceptions. The cells are large, containing several nuclei, which arrange themselves in the center of the cell, differing from the giant cell of tuberculosis, in which the nuclei are arranged around the periphery. Cells of spindle- or round-celled type may also be observed in these sections.

Causation.—No definite causation has been demonstrated. Many of these growths are recognized by the patient after a traumatism has called attention to the part. It is often difficult to determine whether the growth antedated the injury, or the injury preceded the neoplasm. In

one of my own cases such a condition existed, the patient first becoming cognizant of an intra-abdominal mass about three weeks after a moderately severe abdominal traumatism.

Most writers agree that traumatism is not an important factor, yet it cannot be said that this is quite proved.

The writer¹⁰ a number of years ago studied the causation of these neoplasms, and arrived at the conclusion that the cause was a living entity not yet determined. It is fully recognized that this view runs counter to the weight of opinion, but the arguments presented by those holding opposite views at that time have not convinced us that our contention was not tenable. At present this point is not yet finally determined. Some recent observations have been made that seem to confirm the opinion expressed above.

Symptoms.—All of these tumors are persistent in their extension. Even the least malignant form grows progressively, although at times spasmodically. They frequently exist for considerable periods of time before exciting sufficient discomfort to call the attention of the individual to their presence. While in some instances their early development is slow and insidious, they may develop very rapidly, gaining considerable size within a few weeks.

The symptoms depend upon the location of the growth, its rapidity of development and the pressure upon adjacent structures. When pressing upon the nerve trunks or even smaller branches, pain is the first indication shown by the patient. Pressure upon the vessels from the extremity is shown by swelling and edema in the corresponding limb. Interference, even partial, with the movements of the intestine or with the fecal flow may produce cramps or constipation. Some have diarrhea. The latter occurs if the intestinal mucosa is abraded from pressure.

The first symptom presenting may be intestinal occlusion. Jaundice is a rare symptom, and results from pressure upon the bile passages. A prominent late symptom is loss of weight. Cachexia is also present when the diseased condition is sufficiently advanced to interfere with nutrition. Pressure upon the thoracic duct may produce chylous ascites.

In the 28 cases recorded by Andrews, fever was present in nearly one half, ranging from 99 to 102. Of 24 of these cases in which blood examination was made, the hemoglobin was below 70 per cent in 11. In my cases the red cell count was not much impaired until late in the disease. The white count shows only slight actual change. Relatively there is a slight increase.

The symptoms in the absence of intervention progress until the abdomen becomes distended with fluid. Pain is marked and persistent.

Emaciation progressively increases, and the patient finally dies of exhaustion.

The prognosis is essentially grave, most cases progressing without treatment to a rapidly fatal issue. Early recognition puts these sufferers within the operable group of neoplasms. While the mortality and morbidity are high a considerable percentage may be saved.

The diagnosis, after the patient notices his symptoms, can be made with fair accuracy by a close observer. The presence of a demonstrable mass within the abdomen encroaching upon the peritoneal sac from behind, causing pain, and edema at the ankles interfering with the function of the alimentary tract, will call the attention promptly to this condition.

Deep percussion will elicit dullness, with a superficial tympany on light percussion, and point to its retroperitoneal position. If it has pushed the intestines aside and lies in contact with the abdominal wall, it may be palpated. Its characteristic fixation will aid in distinguishing the tumor from an ovarian cyst, a leiomyoma of the uterus (fibroid) or other intraperitoneal mass. Pyelography will determine that it is not of renal origin. Roentgenograms made without and with pneumoperitoneum should aid in clearing up the diagnosis, the latter method being particularly valuable.

Having determined that the growth is retroperitoneal and not renal or attached to the suprarenal structure, it is to be differentiated from caseous tuberculous glands; from retroperitoneal cysts, either serous or chylous; from aneurysm, and from syphilitic adenitis or gumma; cysts of the pancreas, also from Hodgkin's disease, and from abscesses.

Tuberculous caseation of the retroperitoneal lymph-nodes is more frequent than retroperitoneal sarcoma. It will usually be accompanied by other evidence of tuberculous deposits in other organs. The Calmette, von Pirquet, or other reaction may be employed.

Retroperitoneal cysts grow much more slowly, cause much less systemic disturbance and if reaching considerable size will give evidence of fluctuation. The aspirating syringe may be necessary for differentiation.

Pancreatic cysts develop from a rather definite location, push forward above the stomach, between the stomach and colon, or below the colon. There is usually some digestive disturbance early. There may be some localized tenderness, but not much pain in pancreatic cysts, although the first symptoms noted by the patient may be a sudden pain in the epigastrium, which was noticed months perhaps before the mass appeared. This is quite different from sarcoma.

Aneurysm will show its distinct expansile pulsation, with a marked

and characteristic bruit. It must be remembered, however, that a retroperitoneal growth may by pressure on the aorta produce symptoms closely simulating an aneurysm. Only those cases lying directly over the artery will be likely to confuse the observer. Gummata and luetic adenitis may resemble retroperitoneal sarcoma closely. The history, the positive Wassermann, the diminution in size upon active specific treatment will clear up the case. A negative Wassermann is not positive proof of the absence of lues, however. The picture of Hodgkin's disease is so characteristic that it is unlikely to confuse. Examination of the blood, the high leukocyte count and other enlarged glands will make the recognition of Hodgkin's disease positive.

The treatment of retroperitoneal sarcoma is best accomplished by radical excision through the transperitoneal route, when the size of the growth, its attachments, and the condition of the patient justify the effort to remove the neoplasm.

Following the operation and in inoperable cases as well, Coley's⁶ serum, which consists of the active toxins of streptococcus and bacillus prodigiosus, may be used with benefit. Some remarkable results have followed the employment of the Coley treatment in this class of cases. The treatment should be vigorously carried out as soon as it becomes evident from the reaction after small doses that the patient is able to stand the more severe reaction of larger dosage. Coley has been able to show some striking cures while others do not seem to get such favorable results. This is probably due to the fact that Coley applies the treatment vigorously and persistently.

Radium has seemed to act most beneficially in sarcomata, particularly of this region. It should be used by skilled radiologists to obtain its best effects.

X-ray therapy may be used alone or in connection with the previously mentioned procedures, but it will probably not be as beneficial as will radium.

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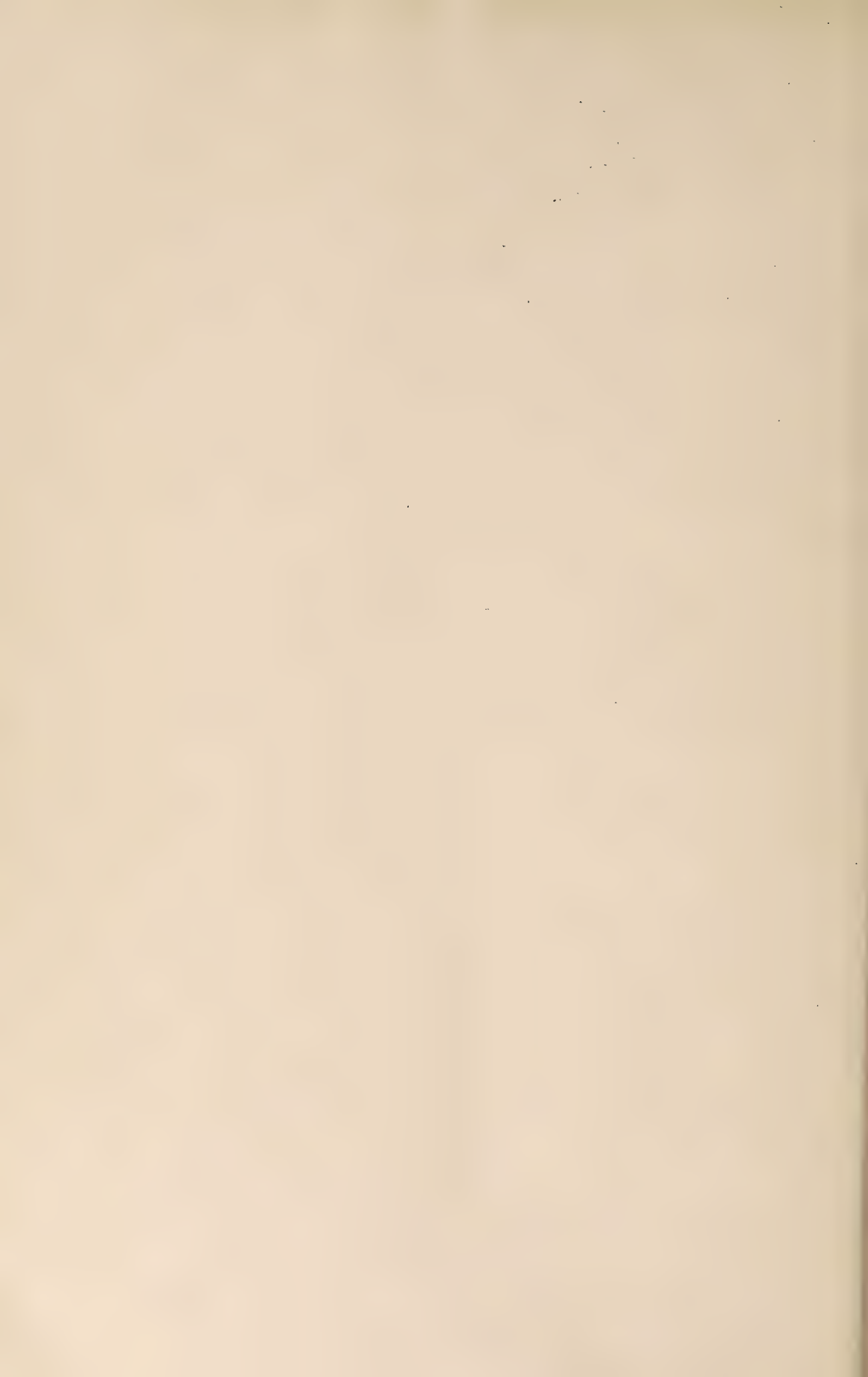
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